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THE
LARYNGOSCOPE.

VOL. LIII APRIL, 1943. No. 4

INTERPRETATION OF HEARING TESTS.*

DR. CHARLES E. KINNEY, Cleveland.

If one were to attempt to digest and assimilate all that has been written about testing hearing it probably would consume the lifetime of an ordinary person; nevertheless, it is the author's hope that this paper might serve to bring our thinking up-to-date. The entire subject is one of confusion and the more we try to bring order out of this state the more confused we become; however, the attempt has brought much pleasure and definite knowledge to the writer, and it may possibly stimulate further work in this field.

As a medical student the whole idea of hearing seemed very simple. There were only two outstanding theories; namely, the so-called "Piano Theory of Helmholtz" and the so-called "Telephone Theory of Central Analysis," commonly associated with the name of Ewald. The only true objection to the Helmholtz Theory was that the basilar membrane is not a series of independent fibres but a continuous layer. Among the supporters of the Central Telephone Theory were many men, but they were practically all anatomists or neurologists. There has been no conclusive new work on the central connection of VIIIth nerve in the past half-century.

In applying the theories of hearing to actual tests on patients who were interested in their own hearing, there were two main methods of testing. One method was directed towards determining the amount of hearing loss; the other was an attempt to locate the lesion that caused the diminution in hearing. For determining the amount of loss there was an

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acoumeter or the watch as a mechanical means and a more demonstrable means, particularly to the patient, was a test by use of the voice — either spoken or whispered. The tuning forks were used to try to determine where the impairment occurred with respect to the range of hearing, whether high pitched, low pitched or intermediate. Since the acoumeter was a foreign instrument, fairly delicate to manipulate, it was not greatly used in this country and the watch tick test and spoken voice test were the methods of determining amount of hearing loss. During the first World War standards for rejection or admission in the Army and Navy were based upon these two tests. It, therefore, became common practice for the otologist to make his estimation of hearing mainly on the spoken voice test because the watch tick test had limitations, particularly in that it was usually a high pitched instrument. In a healing otitis media or mastoid the progress of the return of hearing was usually checked by the spoken voice test. This was also the test of choice in checking the results of opening an Eustachian tube, either by the catheter or Politzer method. The tuning forks were valuable but had the great drawback of no uniform method of excitement and also their very limited length of time in which they remained excited. As a consequence, the tuning forks in the otologist's office were seldom used except on the first examination and then only when the otologist had a particularly perplexing problem. To have said, either in a written statement or before a group of otologists, that politzerization or Eustachian tube catheterization was of no therapeutic value in increasing one's hearing would have been heresy of the first degree.

In the early 20's Bunch and Dean devised an instrument for testing hearing by pure tones with the aid of the radio tube; this was known as an audiometer. Because this instrument could be made to emit essentially pure tones for a prolonged period and the intensity of these tones could be varied at will it soon became an instrument to both supplant the tuning forks as a method of diagnosing the location of a hearing impairment lesion and also the spoken voice test or the watch tick test as an instrument to diagnose the amount of impairment. Naturally there were several types of audiometers then to appear on the market but fortunately the American Medical Association, through its Council of Physi-

cal Therapy, stepped in to formulate acceptable standards for an audiometer. These standards have now been decided upon and practically everyone recognizes these standards. It is interesting to note in passing, however, that the scale on the audiometer was chosen to be the C scale, which corresponded to the majority of tuning forks in use. There was introduced a new unit of sound, namely the bel and its stepchild, the decibel, which is one-tenth of a bel. To exactly define a decibel is very difficult because this unit has a physiological, a mathematical and a physical basis. As near as can be ascertained a bel represents the mathematical power of 10 that is the equivalent of the intensity ratio over that of the minimal audible tone in the average human ear.

As the use of the audiometer became more widespread there were many interesting observations noted and many ideas about some of our therapy were attacked by eminent otologists. One of these statements was to the effect that the otologist who inflated an Eustachian tube and charged the patient for this service was no more than a highway robber. The basis of this statement lay in the fact that the otologist had made a pure tone audiometric examination of a person's hearing and then inflated their tube and could show very little if any change in a pure tone audiometric reading. It was while pondering this question, of a rather serious nature, that the writer was led about four years ago to make the following observation:

A series of 14 consecutive cases of adults who complained of a rather sudden diminution of hearing in one ear was studied. Nine of these patients said the sensation occurred either during or immediately after an airplane trip, three of the cases occurred at the end of an acute cold, and two cases were of no true known etiology. All of these cases were subjected to a pure tone audiometric examination before and after the treatment. The average decibel loss on the pure tone audiometer for the three frequencies of the so-called speech area, namely, 512, 1,024 and 2,048, in the affected ear was 12 dcb. as compared to the patient's unaffected ear. All of these 14 patients had less than five feet of hearing in the affected ear testing with a Bárány noise apparatus in the opposite ear. After a successful inflation of the Eustachian tube on the affected side, pure tone audiometric readings were as follows: In six cases there was no demonstrable change

on the pure tone audiometer. In four cases there was an average improvement in the frequencies mentioned of 6 dcb. In the remaining four cases the pure tone audiometric readings on both sides were essentially the same; however, the patient in every case immediately stated that the hearing was better, the ear felt better, and upon examining by the spoken voice it was found that every one of them had at least 30 feet hearing in the previously poor ear.

This discrepancy in the older and newer method of testing brings up several interesting problems. What is the relationship between one's ability to perceive and accurately identify pure tone and the same individual's ability to perceive, identify and analyze the spoken voice?

About this time there began to appear reports on the improvement of hearing in cases of so-called conduction type of impairment, not of middle ear infectious type. One operation advocated was the production of an additional channel for the transmission of sound waves to the middle ear by making a skin tube in through the mastoid antrum to the middle ear. The other method was by putting a window in the external semicircular canal in order that sound waves might be more easily transmitted to the endolymph. In both instances the surgeons claimed that the person's improvement in hearing was much greater than could be demonstrated on a before and after pure tone audiometric examination. In the writer's experience of two cases where the fenestration of the horizontal semicircular canal operation had been performed by another person this was true. More recently in personal communication with otologists working in the United States Army Air Corps another similar phenomena has been observed; namely, that certain pilots when subjected to the rarefied atmosphere of 30,000 feet or greater complained that their hearing over their radio was not very good. These pilots at first complained that the radio did not work at these altitudes. Checks on those instruments in reduced air pressure chambers revealed there was no difference in the response of the radio. These same pilots were then subjected to the rarefied atmosphere comparable to these high altitudes in a pressure reducing chamber and it was found that their ability to perceive pure tones was not changed, although they did complain of an inability to hear the spoken voice over the radio.

Another phenomenon in testing for pure tones by the audiometer that was noted by the writer is the phenomenon comparable to the after-image of the eye. In many cases it was noted that both children and adults seem to hear a pure tone at a much less intensity as the tone was reduced in intensity than the same person could hear the tone when the intensity was set below their threshold and increased up to the point where the person could hear it. Over 50 years ago Politzer mentioned this same fact with respect to testing by the spoken voice. In a series of 86 consecutive cases of pure tone audiometric examination it was found that this disparity at the frequency of 1,024 amounted to approximately 8 dcb. So far there has been no known attempt to standardize the method of recording one's hearing on a pure tone audiometer, although the manufacturers of hearing aids all advocate the use of the lowest point at which the person hears the tone when the intensity is being reduced. For several years the writer has been following the policy of testing at each frequency by both reducing the frequency down to the minimal audible threshold and then turning the instrument below that and raising it until the person again hears the tone, thus taking an average of these two points. It is suggested that some official body adopt a policy towards this point because many of our audiometric reports do not make this item clear.

The next item that interested the writer was the observation that children with an appreciable bilateral hearing los seemed to hear much better when using both ears. These original observations were made by testing school children in a relatively quiet room with a Bárány noise apparatus held in the untested ear. Not infrequently would one find a case similar to the following:

A 10-year-old child with healed bilateral otitis media, both drums quite scarred and retracted, had hearing in the right ear of 10 feet, the left ear 15 feet. When the child was tested binaurally his hearing was 25 to 30 feet.

An attempt was then made to duplicate these findings on the pure tone audiometer. Two earpieces were introduced into the circuit of the outlet jack and by studies with a sound level meter it was determined that the two earpieces used in the same circuit reduced the output in all the frequencies on

an average of 4 dcb. Sixty-one cases were checked by this method. A pure tone audiometric examination would be made of both ears and then this pure tone audiometric examination was immediately repeated using the earphones on both ears. The findings of this binaural testing were corrected at each frequency. In only four cases out of the 61 was the average binaural hearing 5 or more decibels greater for the six frequencies of 128 to 4,096 inclusive, and in no case was it greater than 10 dcb. Of these 61 cases, 22 showed an average decibel loss in the better ear, for the six frequencies of 128 to 4,096, of 30 dcb. or more. These 22 cases were then checked by the spoken voice method and in every case when tested binaurally the child could hear 10 feet better than in the better ear. In a few instances this was 30 feet better when tested binaurally.

From the above experiences it was concluded that an individual's ability to perceive pure tone is no better when using both ears than in the better ear. By the same token it was also shown that an individual's ability to hear and analyze speech was definitely better binaurally than their ability to hear and analyze speech in the better tear. It was then felt that a true analysis of one's hearing and estimation of their loss, particularly in percentage figures, was not complete unless one made an estimate of the hearing loss to pure tone and also a hearing loss to the spoken voice.

Well realizing the criticism inherent in any spoken voice test by previous methods, an attempt was made to develop a more accurate method of testing in this manner. One of the newer types of audiometers had a microphone circuit included in the set, ostensibly to communicate with the person being tested. It was found that if the source of the voice was kept constant the output in the ear receiver could be varied by regulating the intensity dial. An attempt was made by using a phonograph recording introduced in the microphone circuit. Dr. McFarland, of Philadelphia, had devised a record which had constant intensity output of two digit numbers and two- and three-syllable words varied as to their containing consonants and vowels. This made the apparatus rather complicated, particularly for use in an otologist's office, and the results were not very satisfactory. A series of tests on normal hearing persons was then done to see how constant a

person could keep his voice speaking into the microphone and taking a reading on the intensity dial as to where it was thought the person being tested missed the majority of numbers and names given. It was felt that this was a rather satisfactory method and definitely much simpler than the phonograph. A series of 276 persons were tested both on the pure tone audiometer and for speech through the audiometer by use of the microphone circuit. Complete details of these series of cases and results were published in a previous paper.[1] In this series of cases it was found that with the exception of the child with nerve deafness the decibel loss to speech was less than the decibel loss to pure tones. Thirty-one nerve loss cases in children showed the reverse to be true. The amount varied depending upon the diagnosis. The so-called otosclerotic type were much less impaired to the spoken voice than they were to the pure tone; while in typical cases of otitis media the impairment was nearly the same. From the foregoing experiments it was concluded that any attempt to give a percentage loss of hearing without taking into consideration both the pure tone loss and the voice loss was incorrect. Consequently, the writer then devised a method of mathematical calculation of percentage loss of hearing. This method took into consideration both the patient's ability to perceive pure tone and the voice.

About this time a committee was appointed by the American Medical Association to devise more accurate methods of determining a percentage loss of hearing. Dr. W. E. Grove was chairman of this committee. This committee has recommended to the American Medical Association, and the latter body has adopted, a method of determining percentage loss of hearing based on the phenomenon of weighting the pure tone audiometric findings in order that they might more closely correspond to the known facts about the frequency characteristics of the human speech.[2]

In order to compare the American Medical Association's adopted method of percentage loss calculation with that previously described, a series of 22 cases of bilateral healed otitis media were studied. In each instance the percentage loss as figured by the American Medical Association method was definitely less than by the writer's method. This difference varied from 8 per cent to 22 per cent; the average of the

22 cases was 11.6 per cent less loss by the American Medical Association method than by the writer's method. The minimal loss by the American Medical Association method in either ear of the 22 cases was 6.2 per cent and the maximum loss was 27.2 per cent; in other words, these cases might be classed as moderate bilateral hearing losses, but when one figured the combined per cent loss for both ears by the American Medical Association method this figure corresponded more closely to the actual loss to speech when tested binaurally. Every one of these cases could hear at least 30 feet when tested binaurally.

Nine cases of rather typical so-called otosclerosis were then compared by the two methods. Three of these cases were children in their early teens. The American Medical Association method in these cases showed an average percentage loss of 22 per cent greater than by the writer's method. The reason for this discrepancy is that although these three children had an appreciable loss on the pure tone audiometer they had no loss to voice test; as a matter of fact all three children could hear at least fifty feet in either ear. Four of these nine cases were persons between the ages of 20 to 40 and in every one of these cases the percentage loss was essentially the same by the two methods. The remaining two cases were far advanced, over 40 years of age, and in these two cases the percentage loss by the American Medical Association method was approximately 10 per cent less than by the writer's method. These two cases were ones commonly referred to as being stone deaf.

A series of seven cases of unilateral nerve deafness were then compared by the two methods. Five of these seven cases could not hear a shout in the affected ear, while two of them could make out a loud shout. The average percentage loss by the American Medical Association method of these seven cases was 18.1 per cent less than by the writer's method. All seven of these cases had essentially normal hearing in the untested ear and the discrepancy between the two methods may lie in the fact that the pure tone readings were not as low as they should be because of poor masking in the unaffected ear, although masking was attempted by the usual masking attachments.

In summation, conclusions from the above-mentioned observations can be drawn as follows:

1. Official standardization as to the point of recording hearing decibel loss should be adopted.

2. A person cannot hear pure tones binaurally any better than he can in his better ear.

3. A person can hear the spoken voice better binaurally than he can in his better ear providing there is a bilateral impairment.

4. The American Medical Association's adopted method of determining percentage hearing loss is far superior to any method previously adopted. Because more weight in the determination of the figures was given to the laboratory analysis rather than on clinical cases, the American Medical Association's method is inaccurate in the following cases:

A. Cases of so-called otosclerotic type in younger persons where the pure tone loss is much greater than the voice loss.

B. Cases of unilateral nerve deafness type where the ability to perceive speech is practically nil while they still have some ability to perceive pure tones.

5. While ability to perceive and analyze essentially pure tones may lie completely in the cochlea, ability to perceive and analyze more complicated tone patterns in the form of human speech probably lies more centrally and has a bilateral innervation.

6. More accurate methods of estimating one's ability to perceive speech must be developed before true analysis of hearing loss can be accurately determined.

7. When these better methods are developed and a better understanding of the central connections of the eighth nucleus are made some of the newer therapeutic methods for improving an impaired hearing will be more readily evaluated.

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10515 Carnegie Avenue.

FACIAL PARALYSIS FOLLOWING MASTOID SURGERY. REPORT OF THREE CASES TREATED SUCCESSFULLY.*

DR. JULIUS W. MCCALL and DR. FREDERICK S. GARDINER,
Cleveland.

Facial paralysis following mastoid surgery is a discouraging complication to the aural surgeon and a depressing condition to the patient. Since the remarkable work of the late Charles Ballance and Arthur Duel on this subject, several articles have been written with reported good results. The purpose of this paper is to describe the treatment of postoperative facial paralysis, presenting three cases so treated.

The facial nerve is chiefly a motor nerve supplying the muscles of facial expression. It emerges from the petrous portion of the temporal bone and passes backward along the superior portion of the inner wall of the tympanic cavity just above the oval window. It then courses downward across the inner and inferior wall of the aditus ad antrum immediately below the upper and deeper portion of the horizontal semicircular canal. From there it passes downward, deeply buried in the plate of bone forming the posterior wall of the auditory canal. and emerges just posteriorly to the styloid process, distributing itself to the muscles of expression of the face.

Injury to the facial nerve during surgery for acute and chronic mastoiditis is an undesired complication. The nerve is usually injured as it courses across the inner wall of the tympanic cavity, where it is covered by a thin layer of bone and sometimes only by mucous membrane, at its bend just below the horizontal semicircular canal, or as it courses deeply in the mastoid process. The types of injury are usually compression from edema, packing, blood clots or bone chips, and division from bone chips or instrumentation. The resulting paralysis is of the peripheral type. The movements of facial expression on the involved side are lost, including wrinkling of the forehead. This is an action of the frontalis and is not

*From the Otorhinolaryngological Department of St. Luke's Hospital, Cleveland.

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lost in central lesions of facial paralysis. When division or compression produces injury of a motor nerve, such as the facial, Wallerian degeneration takes place. The axone cylinders in the distal segment degenerate out to the myoneural junction of the muscle, leaving the sheaths or pathways intact. While, in the proximal segment, a part of the central nervous system, the axone cylinders continue to grow, forming a neuromatous bulb. It is this factor of Wallerian degeneration that makes possible the repair of an injured facial nerve by free nerve grafting, which will be discussed later in this paper.

As Collier[2] states: "The treatment of injuries to the facial nerve depends on the type of injury and the anatomical completeness of division. This applies not only to the facial but to any of the peripheral nerves. If the accident has been such that it is likely to interfere with the continuity of the nerve, and if the clinical examination shows total loss of function in the parts supplied, the correct surgical treatment is exploration. This surgical principle was not used in facial paralysis until publication of the work of Ballance and Duel (1934)." Criteria as to type of treatment in these cases is not always easy. An onset of facial paralysis 12, 24 or 48 hours after operation is not likely to be due to a discontinuity of the nerve. An onset of paralysis immediately or a few hours after operation is a different problem. Here the most reliable guide to the anatomical and physiological continuity of the nerve is its response to stimulation by faradic current. A negative response shows an interference with nerve conduction either from division or compression. A positive response indicates an intact nerve which has a physiological block from edema, packing or clot.

When the faradic response has been lost, the treatment is immediate. The mastoid wound should be reopened and any compressing elements removed. If there is division with loss of substance, then a free nerve graft must be done. Where the faradic response is retained, with early or late onset of paralysis, observation with supportive treatment is the procedure. These usually go on to complete recovery. Any packing in such cases should be loosened or removed, and the condition of the nerve tested by faradic stimulation. Galvanic stimulation as well as heat and massage should be applied to

the paralyzed muscles to maintain their tone; however, if at any time the faradic response is lost, then a free nerve graft is to be recommended.

Another type of facial paralysis encountered is one of longer duration. Usually the mastoid infection has cleared up, the paralysis having occurred at the time of surgery. Although the condition is recognized, the surgeon does not attempt to correct it, preferring to rely on electrical stimulation with the hope of restoring function to the paralyzed side of the face. This hope is futile. Good results in many of these cases, if not of too long standing, may be accomplished by a free nerve graft operation.

The criteria for operation in cases of long duration is the reaction of the paralyzed muscles to galvanic stimulation. Where faradic stimulation tests the activity of the nerve, galvanic stimulation tests the muscle. A positive response is the indication for operation. A negative response means that degeneration and probably fibrosis of the muscle has occurred, and it will not respond to stimulation, either nervous or electrical. Ballance and Duel[3] say it is not justifiable to operate if a galvanic response cannot be obtained from the muscles of the face, and in long standing cases the examination should be done under an anesthetic. They state that it is useless to heal the nerve if there are no muscular fibres to energize.

The general treatment of facial paralysis has been referred to above. Through the exhaustive work of Ballance and Duel[3] on facial paralysis, an answer has been found to the treatment of many of these cases. These authors carried out experimental work on Rhesus monkeys in the treatment of facial paralysis by nerve grafting. Their work was as instructive concerning the fate of a nerve graft as it was in regard to the treatment of facial paralysis. A nerve graft, whether sensory or motor, has but one fate, which is degeneration of the axone cylinders while the sheaths remain intact. A freshly cut nerve takes about two weeks to degenerate. In its approximation to the divided nerve the freshly cut cylinders of the proximal part follow down the newly presented pathways of the graft, continuing in the pathways of the distal fragment (see Wallerian degeneration above) to the myoneural junction of the facial muscles. The impulses are then transmitted to the previously paralyzed muscles and they once more resume a functioning activity.

Briefly, their operation consists of three stages: *1.* The mastoid operation, thorough and complete; *2.* operation on the Fallopian aqueduct — here the outer wall is removed, exposing the facial nerve from its course in the middle ear to its exit from the stylomastoid foramen; *3.* the introduction of the nerve graft. With the nerve exposed, the damaged part is cut away and the proximal and distal ends are freshened up. The gap is then filled with a nerve graft taken from either a motor or sensory nerve the size of the damaged portion. A protective covering is placed over the nerve and the wound closed.

The authors stress that the time to operate is when the diagnosis is made. Surgery is recommended at once for the relief of damage to the nerve, if the paralysis has been caused by a recent injury. If the paralysis has been prosent for approximately six months in a patient with chronic otorrhea, Ballance and Duel deemed it advisable to postpone the operation until healthy granulations have formed around the Fallopian canal; however, it was their opinion that the earlier the operation the easier it is to do and the more certainty there is of complete recovery of the nerve. When are results to be expected? Cases with results have been reported as early as three months, but hope should not be given up for two years. Supportive therapy should be administered to keep up the tone of the muscle while waiting for recovery; however, once recovery has begun, these should be withdrawn, especially electrical stimulation, as this may give an overcontracture of the newly functioning muscles.

Report of three cases of facial paralysis following mastoid surgery and their treatment resulting in a cure in all three cases:

Case 1: E. H., female, age 48 years. Diagnosis: Aural polyp, chronic otitis media, chronic sclerotic mastoiditis (right). Operation: Right radical mastoidectomy. At the operation, while examining the stalk of the aural polyp in the middle ear with a dull tip finder, contraction of the facial muscles was noticed by the anesthetist. This was due to an exposed nerve resulting from the chronic infectious process. At the conclusion of the operation there was no evidence of injury to the facial nerve. On regaining consciousness the patient had full function of the right side of her face. The hospital course was uneventful up to the eighth postoperative day. On this day there was an onset of right-sided facial paralysis (see Fig. 1A). With faradic stimulation the nerve responded positively. Diagnosis: Physiological nerve block, nerve intact. Galvanic stimulation, heat and gentle massage were applied to the paralyzed muscles every other day. The faradic response remained positive and 16 days after the

onset of the paralysis there was beginning function of the paralyzed muscles. Their activity continued and in about five days they were normal and equal with the opposite side of the face (see Fig. 1B). On the

Fig. 1A. Facial paralysis occurring eight days after radical mastoidectomy. Note complete paralysis of the right side of face.

Fig. 1B. Resumption of activity of the paralyzed muscles 16 days after onset of the paralysis.

last visit, which was four months postoperative, the ear was dry and all the facial movements were normal.

Case 2: J. R., male, age 38 years. Diagnosis: Chronic otitis media,

chronic mastoiditis (right). Operation: Right radical mastoidectomy. At the conclusion of the operation and upon recovery from the anesthetic there was evidence of facial paralysis. Four days postoperative, the patient was taken to surgery and the facial nerve was uncovered for 1 cm. from its course in the middle ear to its disappearance deep in the facial ridge. Two depressed bone chips in the facial canal were found causing pressure on the nerve, opposite the horizontal semicircular canal. The bone chips were carefully removed. During and after his hospital course, galvanic stimulation, heat and massage were applied to the paralyzed facial muscles. Approximately five months after decompression slight movements returned to the right side of the face. These continued to improve until the movements of both sides of the face were about

Fig. 2. Recovery from facial paralysis following decompression of right facial nerve.

equal except for the right frontalis muscle, which never returned. Diagnosis: Compression of the facial nerve resulting in degeneration. Regeneration following removal of the compression elements (see Fig. 2, taken three years later).

Case 3: W. L., male, age 7 years. Diagnosis: Acute mastoiditis. Operation: Simple mastoidectomy. Right-sided facial paralysis occurred following operation. We did not see this case until 18 months after the onset of the paralysis. There was no response to Faradic stimulation of the facial nerve; however, we obtained a response of the facial muscles to galvanic stimulation. Operation: Free nerve graft by the method of Ballance and Duel. Under general anesthesia the old mastoid wound was reopened. The facial canal was exposed and the facial nerve was found to be severed about 2 mm. below the horizontal semicircular canal, with loss of three-quarters of a centimeter of the nerve. A neuroma on the proximal end of the severed nerve was removed, and this as well as the distal end was freshened up. Next, the nerve graft was obtained by exposing the lateral femoral cutaneous nerve. A section was removed, the size and length of the severed portion of the facial nerve. This graft was placed in the bony canal approximating the freshened proximal and distal ends of the open facial nerve. The nerve graft was covered with

a Penrose drain and the wound closed. During his hospital course, and after, galvanic stimulation, heat and massage were applied to the paralyzed muscles. Nine months later the child's mother observed that the boy was able to move the previously paralyzed corner of his mouth. Gradually the paralyzed muscles, with the exception of the frontalis, have assumed a functioning activity. Diagnosis: Degeneration of the facial nerve following division and loss of nerve substance. Regeneration secured by the free nerve graft operation (see Fig. 3).

The above cases represent three types of facial paralysis. The first, a physiological block of the facial nerve; the second, a compression of the nerve by a bony fragment resulting in degeneration of the nerve with regeneration after the compressing element was removed; and third, a division with loss of nerve substance, regeneration procured by nerve grafting.

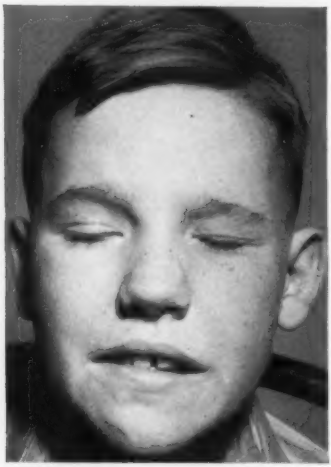

Fig. 3. Recovery from facial paralysis. Cause: division and loss of nerve substance. Treatment: the free nerve graft operation outlined by Ballance and Duel.

In cases where regeneration follows degeneration the frontalis muscle remains paralyzed. This was true in our cases (see Cases 2 and 3). It is reported that associated facial movements do sometimes occur following regeneration of the nerve. For example, winking of the eye will sometimes occur with movement of the corner of the mouth, a condition usually overcome by re-education of the muscles. This did not happen in any of our cases.

Prior to the work of Ballance and Duel much stress was laid on the facial-anastomotic operations for facial paralysis following mastoid surgery. This type of operation consists of anastomosing one of the cranial nerves, such as the hypoglossal or spinal accessory, to the facial nerve. The impulses of the anastomosed nerve then stimulate the facial muscles. For example, in the case of the spinal accessory nerve, shrugging of the shoulder produced contractions of the face. Although this procedure restored movement to the facial muscles, they were purely unemotional responses. These operations, although still done by some, are outmoded by the Ballance and Duel operation which restores the normal function of the facial nerve and muscle; *i.e.*, the proper emotional response to the central nervous stimuli.

SUMMARY.

1. Facial paralysis following mastoid surgery and its treatment have been discussed.

2. Three cases successfully treated have been reported.

3. Any facial paralysis coming on at the time of or immediately after mastoidectomy demands immediate exploration of the operative area and exposure of the facial nerve.

4. The work of Ballance and Duel has revolutionized the treatment of postoperative facial paralysis.

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CANCER OF THE NOSE AND NASAL SINUSES.*

DR. LEROY A. SCHALL, Boston.

Introduction: The nose and accessory nasal sinuses may be the site of not only primary malignant disease but may be involved by secondary extension from adjacent structures or in rare instances by metastasis from distant lesions.

Thus a growth originating on the eyelid, the lateral external nose, the alveolus or the hard palate may invade and secondarily involve the accessory sinuses. A lesion originating in any of the sinuses may by extension involve other sinuses. Hypernephroma of the kidney and Ewing's tumor of the ilium has been complicated by metastasis to the nasal sinuses.

Symptoms: When a new growth is limited to the nasal cavity, nasal obstruction and hemorrhage are the most frequent symptoms. A small growth of the anaplastic type with thin-walled blood vessels may show early ulceration with subsequent hemorrhage as its first symptom. With the increase in size of the growth, nasal obstruction occurs. This obstruction is usually unilateral, so it is an axiom that any unilateral obstruction by a growth should be considered malignant until the microscope proves it otherwise. When the tumor is of the slow-growing variety, with well developed connective tissue and thick-walled blood vessels, hemorrhage may not occur and the only early symptom may be that of nasal obstruction. The growth may have the appearance of a simple nasal polyp, yet upon removing it the hemorrhage may be excessive; and this should be suggestive of cancer. It hardly seems necessary to say that every specimen should be examined microscopically.

The symptoms of new growth of the maxillary sinus depend somewhat upon the location of the growth. Growths originating in the hard palate or the superior alveolus are characterized by *early swelling.* In the early phase there is but little pain. With the invasion and destruction of the bone, the teeth may loosen and fall out, but it is a comparatively pain-

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less process. Extensive ulceration may develop with fistulization into the maxillary antrum and still be painless. A patient under my care states that the first symptom he noticed from a lesion starting in the hard palate was the fact that upon drinking, liquids came through his nose.

Lesions originating from the posterior, superior antral wall are characterized by a parathesia or an anesthesia of the cheek of that side. They are ofttimes accompaned by pain referable to the teeth. It is not unusual for these patients to seek relief by repeated dental examinations and by the loss of one tooth after another in a vain effort to alleviate the discomfort. As infection is superimposed upon the original lesion, there may be early pain referred to the cheek; and puncture and lavage of the antrum has been carried out in many cases. With the increase in size of the new growth, there is extension into the nasal cavity with a mass which may block the entire nostril. Then, too, there is extension and destruction of the bones of the superior maxilla, with erosion of the anterior face of the maxillary antrum, or destruction of the rim of the orbit. Many patients do not seek medical advice until the growth has reached such a size that there is a mass over the cheek, or until the extension into the orbit has produced an exophthalmos.

New growths originating in the posterior part of the ethmoidal labyrinth may extend into the sphenoidal sinus; and if the optic nerve is involved the first symptom is a diminution of vision on that side. Repeated hemorrhage should always be suggestive of cancer. The bleeding may be but a few drops, or a hemorrhage of such intensity that transfusions are necessary.

Biopsy: I am an advocate of biopsy in every case, for our treatment depends upon the type of tumor as shown by the microscopic examination. When in doubt, I am an advocate of an exploratory operation. A lateral rhinotomy or an external canine fossa operation on the maxillary antrum, in my opinion, in a doubtful case is not only advisable but justifiable.

X-ray: A unilateral mass obstructing a nasal cavity is suggestive of cancer. The usual X-ray picture of sinus cancer is that of the late stage, when destruction of bone has already taken place. Repeated X-ray examinations in every

suspicious case should be the rule, if we are going to make the diagnosis early.

Treatment: When there is superimposed infection, adequate drainage must be provided irrespective of what type of treatment is employed. Even in an anaplastic type of growth it may be necessary to provide not only intranasal drainage but drainage by way of the canine fossae, either before, during or after external irradiation. The patient with good drainage has less pain and discomfort during the treatment.

The rational of surgery for malignant disease of the sinuses may be only that of facilitating adequate drainage.

Should the growth be of the anaplastic variety, irradiation, either external or interstitial, should be the treatment of choice. There should be no quarrel with a therapeutic test of irradiation, with the patient under close observation. Should the lesion show regression the treatment can be pushed to the limit of tolerance; however, if no regression is evident after moderate irradiation, particularly in a lesion of the adult or slow-growing type, then surgery plus irradiation is indicated.

Operation for Nasal Lesions: Adequate exposure should be obtained for any operative procedure. New growths limited to the nasal cavity can be adequately exposed through a lateral rhinotomy. This incision starts opposite the inner canthus of the eye and goes down the lateral wall of the nose and around the ala. The periosteum and soft parts are elevated from the bone and the nasal cavity entered by removal of the ascending process of the superior maxilla and the lateral nasal bone. By extending the incision upward, and the bone removal to include the lacrimal bone, access to the frontal sinus can be obtained. This exposure permits access to the ethmoidal region, to the sphenoid, to all of the turbinates; and through this exposure the antrum can be explored. After removal of the new growth, the cavity is lined with vaseline gauze, packed with narrow-folded strips of gauze, in which radium in the form of either platinum or steel needles can be embedded. An attempt should be made to obtain at least 1 cm. distance between the septum and the radium. The septum can be further protected by a shield of at least 1 mm. of lead. The packing is brought out the nostril and the inci-

sion closed, unless it is desirable to leave it open for further inspection.

Tumors of the Sinuses: The surgical approach to tumors of the sinuses should depend upon the point of maximum involvement. If exophthalmos exists, and if it is questionable whether or not the orbit has been invaded, the ideal approach is through the orbit by an exenteration of the orbital contents. This approaches the maxillary antrum from above and the ethmoidal labyrinth from the side. By removing the floor of the orbit, the antrum can be explored; and by removing the lateral orbital wall, the ethmoidal labyrinth can be exenterated. It permits, too, an easy access to the frontal sinus. When the lesion has originated in the superior alveolus, or from the hard palate with extension into the antrum, the ideal approach is from below by resection of the superior alveolus, including half or all of the hard palate. This approach permits access to all the accessory sinuses. It provides the best of drainage, but it handicaps the patient in that he must wear a dental prothesis when eating. Lesions involving the ethmoidal labyrinth and the maxillary sinus without extension into the orbit, or without evident involvement of the hard palate, can be reached by any of the face-splitting operations.

For extensive sinus involvement I have been using a modified Moure incision. This incision starts opposite the inner canthus of the eye, goes down the lateral wall of the nose, around the ala to the midline of the upper lip. It then goes straight down the columella dividing the upper lip and with an incision through the buccal mucous membrane from the nasal spine to the last molar tooth. The entire cheek is turned back, exposing the superior maxilla. This exposure permits excellent inspection of the operative field, and permits the removal of the entire superior maxilla, if it is found to be necessary.

The incision is made with the cutting electric current, and all bleeders are stopped by electrocoagulation. The bone is removed with Rongeur forceps. The tumor is shelled from the antrum by a curette. Its removal is accompanied by a profuse hemorrhage, so that it should be removed rapidly. A large sponge soaked with hot saline is inserted into the cavity and this soon stops the bleeding. Ligation of the carotid

artery has been advocated to control the hemorrhage and to diminish the blood supply of the tumor. I have not found it necessary to ligate the carotid artery in any case. DaCosta quotes 56 different anastomoses of the carotid artery, so that the effects of ligation of the carotid artery last but eight to 10 days.

The ideal removal of any cancerous growth is by its removal through normal tissue. This is absolutely impossible in a region such as the nasal sinuses. Here the removal amounts to a curettage. The operation is ideal for disseminating the growth and producing either distant metastasis or local recurrences. For that reason, the entire operative cavity should be desiccated with the coagulating current. For this reason, too, I have advocated the local insertion of radium to destroy any scattered tumor cells. The orbit is protected with a shield of 1 mm. of lead, the cavity lined with vaseline gauze, gauze packing is inserted and the radium placed within the gauze packing so that there is at least 1 cm. of gauze to act as protection. This gauze is brought out and partly through the buccal incision. The musculature of the lip is sutured with buried catgut sutures, taking care that the vermilion border of the lip is approximated exactly. The skin edges are then closed with dermal sutures. Depending upon the sensitivity of the tumor, the radium is left in place for a total dosage of 2,000 to 4,000 mg. hours. At the end of the desired time, under evipal anesthesia, the packing is removed through the buccal incision. In the after-treatment, care must be taken that the buccal incision does not close. This can be kept open by an obturator made of dental compound.

I am greatly indebted to Ohngren for the suggestion that the odor following this operation can be greatly alleviated by the use of a potassium permanganate spray.

Exophthalmos due to radiation reaction occasionally occurs. It is exceedingly painful, and plastic procedure on the lids to preserve the cornea have been of no avail. This reaction may take place early, or may be delayed as long as six months t- one year after radiation. It is a trying situation, as one must think of orbital extension or recurrence within the orbit. Orbital exenteration may be necessary, not only to relieve the pain but to make certain that extension of the disease has not

taken place. Studies of five such cases show the pathology to be that of edema and fibrosis.

The after-care consists of keeping the cavity clean. The patient can use a nasal irrigation at home with frequent applications of potassium permanganate spray — 1-10,000. The discharge ceases in about two to three months. By keeping the buccal incision open, inspection of the operative cavity can be thoroughly made.

This operative procedure of primary closure of the face incision leaves the patient with a minimum of deformity, and permits the social and business life of the patient to continue without embarrassment.

Conclusion: Experience has taught that cancer of the nose or accessory sinuses is not a hopeless condition. Its treatment calls for courage, both by the physician and the patient. It need not be disfiguring. Too much emphasis cannot be placed on early diagnosis, and a plea for a thorough examination of every patient.

243 Charles Street.

MULTIPLE SPINDLE-CELL SARCOMAS OF THE NECK.*

DR. HANS BRUNNER, Chicago.

There are two types of fibromas of the neck: the superficial and the deeply situated. Superficial fibromas are assumed to originate in the sheaths of the nerves of the skin or in the walls of the blood vessels. This paper is concerned only with the deep fibromas of the neck.

According to de Quervain[1] the deep fibromas are classified as follows: *a. Fibromas of the lateral and posterior part of the neck.* These tumors usually originate in the ligamentum nuchae or in the ganglia of the sympathetic nerve or in the dura of the cervical cord. *b. Fibromas of the anterior part of the neck* which are separated from the above-mentioned tumors by the sternocleidomastoid muscle. It is assumed that these tumors originate in the sheaths of the great blood vessels in the area of the larynx or in the area of the hyoid bone. Among the 25 cases reported by de Quervain there were only two cases of this type. *c. Fibromas situated beneath the sternocleidomastoid muscle.* These are believed to originate from the sheath of the great blood vessels or the great nerves of the neck.

All these types of fibromas grow slowly and remain stationary for a period of years. The period of slow growth, however, may suddenly turn into a period of rapid growth, the tumor occasionally reaching the size of an infant's skull. That rapid growth does not necessarily indicate a sarcomatous transformation of the tumor, although de Quervain,[1] as well as Veau,[2] emphasize that these fibromas not infrequently change into sarcomas.

Since recurrences do not occur in simple fibromas, one must assume that fibromas of the neck which are followed by recurrences were impending on malignancy at the time of extirpation.

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*From the Department of Laryngology, Rhinology and Otology, University of Illinois College of Medicine, Chicago.

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The following case of fibroma of the neck differs from the cases described up to the present in many respects.

CASE REPORT.

T. P., aged 40 years, entered the Illinois Research and Educational Hospital on Dec. 14, 1936. He complained of a cough which developed a

Fig. 1. X-ray picture taken before the removal of the retropharyngeal tumor. Note the overflow of barium into the larynx.

year before. About seven or eight months previously, a swelling appeared in his neck and at the same time he began to have difficulty in swallowing. This became progressively worse and eventually the patient was able to swallow only liquids. In the last few months he lost 13 pounds in weight.

At examination a diffuse, firm and smooth swelling on either side of the neck was found. This swelling moved on swallowing, was not fixed and was only slightly tender. The Kahn test was 4+ positive. X-ray examination with opaque meal showed no obstruction, but there was slight temporary irregular stasis in the cervical portion and an increase of the prevertebral soft tissue shadow in that area. He was dismissed with the diagnosis of tertiary lues.

On Jan. 7, 1937, a tumor was found in the posterior wall of the pharynx, extending toward the base of the tongue, which did not permit the inspection of the larynx. The tumor was of firm consistency and there was no fluctuation. Subsequently the neck tumor gradually grew. The patient was readmitted to the hospital on March 24, 1938, complaining of difficulty in breathing and hoarseness. Examination revealed a large perforation of the nasal septum and of the palate. On March 24, a biopsy was taken from the tumor of the neck (see Fig. 3, I, II), the microscopic exam-

Fig. 2. X-ray picture taken after the removal of the retropharyngeal tumor.

ination of which revealed a hyalinized fibroma containing few degenerated muscle fibres and several perivascular infiltrations consisting of lymphocytes. In the succeeding months the neck tumor grew larger and the dyspnea gradually became much more pronounced; however, there was very little cyanosis. He was readmitted on Jan. 2, 1939, and a tracheotomy was performed. The Wassermann in the blood was negative following an intensive course of antiluetic treatment.

Examination on Feb. 14, 1939, revealed a tumor the size of a grapefruit, in the left half of the neck, compressing the larynx and pushing the trachea markedly forward (see Fig. 1). The boundaries of the tumor were not sharp. The left sternocleidomastoid muscle was pushed outward, but at no place was the muscle perforated. The tumor had a firm consistency and was movable to a slight extent. Occipitalward from the tumor the

carotid artery could be felt. The tumor was not connected either with the mandible or with the thyroid cartilage. Few hard glands were found bilaterally. Inspection of the mouth revealed that the tumor in the posterior wall of the pharynx almost touched the base of the tongue.

Under avertin and local anesthesia an incision was made along the left sternocleidomastoid muscle, extending from the

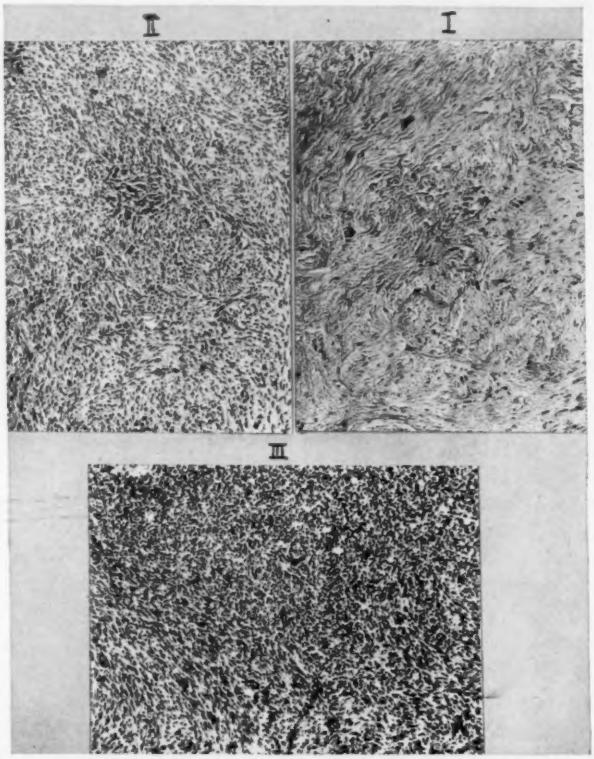

Fig. 3. The pictures I and II present different portions of the retropharyngeal tumor. Picture III presents the structure of the esophageal tumor.

clavicle up to the tip of the mastoid process. A cross incision over the thyroid cartilage was added. The sternocleidomastoid muscle was slit and the omohyoid muscle crossing the inferior part of the tumor was cut through. The sheath of the great blood vessels, situated behind and lateral from the tumor, was separated but not opened. The superior portion

of the tumor was crossed by the hypoglossal nerve. The tumor compressed the submaxillary gland and the left lobe of the thyroid and pushed the entire thyroid gland forward. Upward the tumor reached the inferior margin of the mandible, downward it entered the posterior mediastinum and a pedicle of the tumor extended into the right half of the neck. Several lymph glands and a thick layer of connective tissue covered the tumor, which was connected by connective tissue with the pharynx and esophagus. The tumor was separated from both organs, from the vessel sheath and from the prevertebral fascia by dull dissection. At the moment the tumor was removed, a vasomotor collapse occurred, from which the patient recovered in a few minutes. Drains were put into the retropharyngeal space, and the skin as well as the sternocleidomastoid muscle was partially sutured. The removed tumor had a weight of 345 gm.

During convalescence a lymph stasis of the neck and of the face was noticed; was more marked on the right than on the left but subsided after several days. On Feb. 25, 1939, direct laryngoscopy showed a markedly thickened mucosa throughout. The motility of the right vocal cord was somewhat limited but it moved quite satisfactorily during respiration and phonation. The left vocal cord was fixed in the midline and remained in that position. The wound closed after several days, and after a few weeks the tracheal cannula could be removed (see Fig. 2). The opening in the trachea closed spontaneously and in that stage the patient was presented before the Chicago Laryngological Society on March 4, 1940, by Dr. L. Z. Fischman.

The microscopic examination of the tumor (see Fig. 3) revealed a nodular structure. The bulk of the growth presented every gradation from highly fibrous, acellular tissue through more cellular spindle-cell types up to very cellular structures, with sharply intertwined fibres and nuclei and few blood vessels. Some of the capillaries presented an endothelial proliferation and they were surrounded either by edematous or hyaline connective tissue or by tumor cells resembling a perithelioma. There was a rather sharp margin between the cellular and acellular portion of the tumor. The cells of the tumor were rather uniform, the nuclei rich in

chromatin and there were scanty mitoses. There was a definite fibrillary ground substance even in the cellular part of the growth; however, no perivascular infiltration and no muscle fibres could be seen. Multinuclear giant cells were observed not infrequently.

The patient did not receive postoperative radiation. He was in good health until July, 1942, when he quit his job at a paint factory because he had chest symptoms. He had a bad cough, which, however, improved after he left his job. In September, 1942, the patient began having difficulty in swallowing large pieces of bread or meat. This condition became slightly worse. On Oct. 3, 1942, I saw him again. He complained of slight difficulty in swallowing and breathing. There was no swelling on the neck but there was a firm, immobile gland in the left supraclavicular groove. The scar on the left side of the neck was markedly retracted. There was a pale edema of the lateral wall of the pharynx on the left side extending into the left aryepiglottic fold. The vocal cords were slightly red and edematous and the left vocal cord was fixed but not atrophic. On account of the edema of the pharynx, a direct examination of the larynx was not performed.

On Oct. 4 he had emesis, coughing up about 100 cc. of blood and a piece of tissue. Dyspnea was still present, although apparently not increased. A luetic aneurysm of the aorta was considered. On Oct. 5 he had great difficulty in breathing, cyanosis, and sank into unconsciousness at midnight. Emergency tracheotomy was started but the patient expired before more than the skin had been incised.

The microscopic examination of the piece of tissue coughed up on Oct. 4 revealed a spindle-cell sarcoma. There was an enormous number of nuclei varied in form and size, and rich in chromatin. The fibrillary ground substance was poorly developed. There were many capillaries, several of them filled with hyalin thrombi. In many places the sarcoma cells presented an arrangement typical of a perithelioma. There were necrotic spots with an accumulation of bacteria and there was an intense acute inflammation with production of much fibrin.

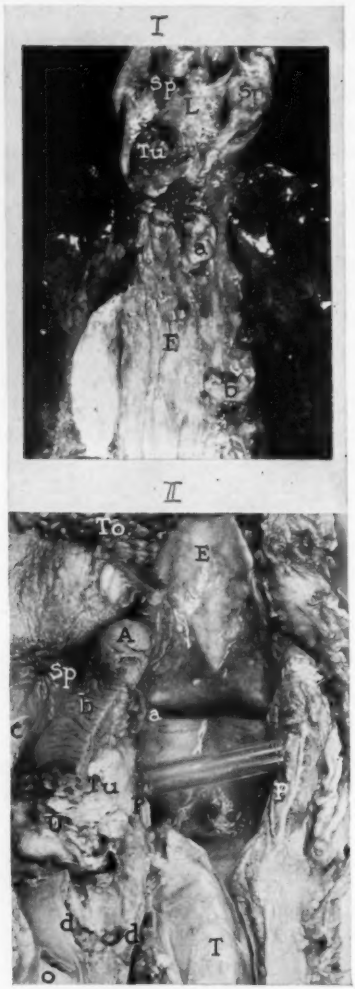

Fig. 4. Picture I presents the anterior wall of the esophagus (E) and the posterior wall of the larynx (L). (Tu) Tumor in the anterior wall of the pharynx; (Sp) sinus pyriformis; (a) and (b) portions of the tumor loosely attached to the anterior wall of the esophagus. In picture II the cricoid plate (P) is cut through. (To) Base of the tongue; (A) edematous mucous membrane of left arytenoid cartilage; (E) epiglottis; (Sp) sinus pyriformis; (a) hernia in the left ventriculus Morgagni; (b) anterior wall of esophagus; (c) posterior wall of esophagus; (Tu) tumor; (U) ulcerated area of the tumor; (d) capsule of tumor; (o) esophagus; (T) trachea.

At autopsy, which was performed by Dr. C. C. Mason, the following findings were reported:

Blood in the trabecular bronchial tree, a patchy bronchopneumonia, a pulmonic congestion of the lower lobes bilaterally, multiple ulcer defects of the stomach, a prostatitis.

Most interesting were the findings in the neck. Beneath the scar on the left side there was considerable fibrous tissue reaction extending over the lateral aspect of the thyroid gland and throughout the region of the sternocleidomastoid muscle. Part of the muscle has been removed. Two cm. below the inferior line of the thyroid cartilage there was a bulging, soft mass of tissue along the left lateral aspect of the trachea and esophagus. The trachea had been pushed to the right by this bulging mass, which extended for a distance of 5 cm. caudad. The tongue, larynx and trachea were removed intact with the lungs and the esophagus.

The esophagus was opened along the posterior aspect, exposing a large ulcerated mass which was situated in the anterior wall of the esophagus and projecting into the lumen (see Fig. 4). This was approximately 3 cm. below the junction of the esophagus and trachea. In spite of the bulging mass, the anterior wall of the esophagus was not stretched and presented circular folds. The tip of the pearlike tumor mass reached the space between the arytenoids, while the inferior pole extended downward about 3 cm. below the inferior border of the cricoid plate. The tumor was separated from the left half of the cricoid plate by a membrane consisting of the perichondrium and the atrophic cricoarytenoid posterior muscle, while the right half of the cricoid plate had no connection with the tumor and was covered by a normal cricoarytenoid muscle. So far as could be seen, neither half of the cricoid plate was invaded by the tumor nor were the muscles between the arytenoid cartilages. Close to the inferior pole of the tumor there was an ulceration in the anterior wall of the esophagus, the size of a quarter of a dollar, which had sharp margins covered with a squamous epithelium and was filled with necrotic tumor masses. Fragments of necrotic tissue had broken away from the ulcerated area and were loosely confined within the lumen of the esophagus (see Fig. 4). Where these necrotic masses came in contact with the posterior wall of the esophagus, the wall had a firm consistency and presented several white, irregular spots. Microscopically, the posterior wall consisted of connective tissue containing several degenerated muscle fibres but no glands. The wall was covered by squamous epithelium. There was no ulceration and no downward growth of the epithelium, but there was a marked subepithelial inflammation.

The tumor had not eroded into the trachea. The inferior pole of the tumor was situated within the muscular coat of the anterior wall of the esophagus and not between esophagus and trachea. The mass of tissue was soft, caseous and measured 4 cm. in its greatest diameter.

The entire larynx was pushed to the right side by the tumor. There was a prolapse of the left ventriculus Morgagni. In the anterior commissure there was a small ulceration. The mucous membrane over the left arytenoid as well as the left epiglottic fold was edematous. The left sinus pyriformis was enlarged but did not present an infiltration by the tumor. There was a bloody red discoloration of the mucosa of the trachea but there were no defects in the mucosa. In the upper part of the trachea there was a longitudinal scar after the tracheotomy.

As already mentioned, the tumor proved to be a spindle-cell sarcoma, parts of the tumor being destroyed by recent bleedings, although in general the growth did not contain many blood vessels. The tumor presented almost the same structure as the one which had been removed by operation (see Fig. 3); however, there were some differences, as the

tumor of the esophagus contained many mitoses, the fibrillary ground substance was almost absent, and there was no definite uniformity of cells. Occasionally large cells resembling ganglia cells could be observed. These cells consisted of a homogeneous protoplasm and contained one or two large nuclei, eccentrically situated, poor in chromatin and containing a relatively large nucleolus. Aside from the ulcerated area, the tumor was separated from the mucous membrane as well as from the cricoid plate by a capsule consisting of a hyalinized connective tissue and several degenerated muscle fibres. At several sites the internal layers of the capsule were infiltrated by the tumor. The mucous membrane covering the tumor was definitely atrophic. It consisted of edematous connective tissue without or with few atrophic glands. The muscularis mucosae was replaced by several degenerate muscle fibres which were scattered throughout the membrane and surrounded by a capsule of connective tissue. The mucous membrane was covered by a squamous epithelium layer.

Summarizing, there was a spindle-cell sarcoma, situated in the external layers of the anterior wall of the upper third of the esophagus, surrounded largely by a capsule and perforating into the lumen of the esophagus in a circumscribed area.

COMMENT.

A luetic individual, 40 years old, developed a tumor of the neck, which was situated between the pharynx, esophagus on one hand, the vessel sheath and the prevertebral fascia on the other hand, extended particularly into the left part of the neck, while a pedicle of the tumor, crossing the posterior wall of the esophagus, extended into the right half of the neck. Concerning the type of the tumor, here was clinical as well as surgical evidence indicating a benign neoplasm. The following findings concurred with that concept:

a. There was no cachexia although the nourishment of the patient was not satisfactory on account of his difficulty in swallowing.

b. The tumor could be removed by dull dissection. There was no infiltration or perforation of the surrounding organs; the esophagus, the vessel sheath and the muscles were simply pushed aside.

c. The tumor was encapsulated.

d. A nodular structure of the tumor was hinted.

e. There were no metastases in a period of at least three years.

f. In three years following the operation no recurrence of the tumor was observed. (This statement does not take into consideration the tumor within the anterior wall of the esophagus, to be discussed later.)

g. The tumor presented a relatively slow growth. According to the history three years elapsed from the time the patient first noticed the tumor until the operation. It is obvious that the period between the actual onset of the tumor and the operation was even longer, as one reasonably can assume that the patient could not notice a retropharyngeal growth unless it interfered with swallowing or produced a swelling of the neck. To give these symptoms, the tumor had to be of considerable size. There was sufficient clinical and surgical evidence to make the diagnosis of a benign tumor and, as a matter of fact, the biopsy taken one year prior to the operation revealed a simple, hyalinized fibroma. The microscopic examination of the removed tumor, however, did not conform with the diagnosis obtained from the biopsy.

The pieces taken from the surface of the tumor revealed the typical structure of a fibroma; however, the examination of other parts of the tumor, particularly those situated close to the center, led to the definite diagnosis of a fibrosarcoma, although the clinical observation did not concur with the diagnosis of a malignancy. During the operation I had the impression that the tumor originated in the loose prevertebral connective tissue. That concept was apparently not correct as "it appears that mesoblastic cells once having been assigned to simple supporting functions have little tendency to neoplastic growth. Many forms of spindle-cell sarcoma prove on closer investigation to belong to the group of tumors of nerve structures, muscle, fat tissue, bone, blood vessels and synovial membrane" (Ewing[3]). In agreement with that statement, Roesner[4] claimed that the retropharyngeal fibromas and fibromasarcoma as well as the fibromas of the neck spring to a great extent from the sympathetic nerve or the

supreme sympathetic ganglion and belong to the entity of von Recklinghausen's neurofibromatosis.

It seems that the statement of Roesner[4] is too specific. The tumors originating in the sympathetic ganglia of the neck, *viz.*, the ganglioneuromas, are situated behind the sheath of the great vessels and push the internal carotid artery forward toward the angle of the mandible, as emphasized by this writer. In the present case, the vessel sheath was situated behind the tumor. This is not in agreement with the findings in growths originating from the sympathetic nerve. This statement, of course, does not imply that the tumor in our case was not of neurogenic origin. On the contrary, the varying proportion of long and short spindle cells lying in a fibrous matrix, both cells and fibres exhibiting marked fasciculation, the sharply intertwined groups of cells and fibres suggest the neurogenic origin of the tumor, although not necessarily of sympathicogenic origin. As in the removed tumor neither nerve fibres nor ganglia cells could be found, there is no strict evidence forthcoming that the growth was a neurofibroma. The same difficulty was met with in the cases of retropharyngeal fibromas described by Semon,[5] New,[6] Albright[7] and others; nevertheless, these authors consider the probable neurogenic origin of the tumor in their cases.

The most conspicuous feature in the present case is, however, the tumor of the esophagus which led to the fatality. This tumor had essentially the same structure as the tumor removed at the operation, except that the great amount of mitoses, the poorly developed fibrillary ground substance and the enormous number of nuclei indicated a higher degree of malignancy. The growth was situated at the junction of the pharynx and esophagus in the anterior wall, did not perforate into the trachea or larynx, but perforated into the esophagus and was encapsulated to a great extent.

The tumor in the case described is interesting because it is rare. There are only somewhat more than 50 cases of sarcomas of esophagus on record (Orton,[8] Dvorak[9]). The growth in this case does not differ essentially from the majority of sarcomas found in the upper third of the esophagus. Several of these tumors are classified as leiomyosarcomas, although there is not always sufficient reason for this classification, as pointed out by Mallory.[10,11] We were not able to decide

whether the tumors in the present case were of myogenic or neurogenic origin.

Of greater interest, however, is the finding that the sarcoma of the esophagus was combined with another fibrosarcoma of the neck, which was situated between the esophagus and vertebra, had no definite connection with the wall of the esophagus and was removed by surgery (see Fig. 5). Thus, the question arises whether or not there is a relationship

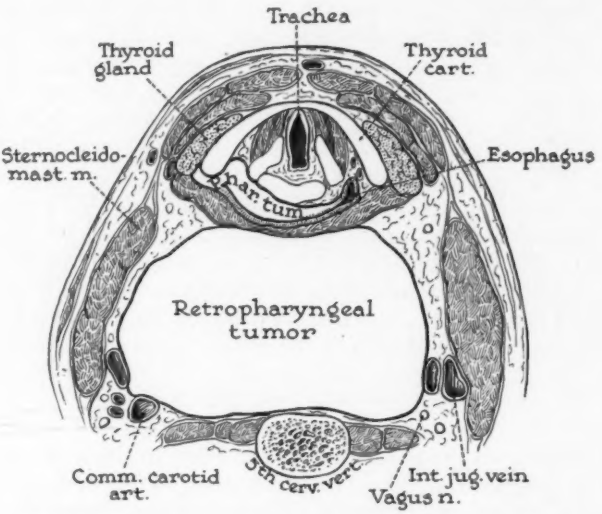

Fig. 5. Diagram presenting the localization of both tumors.

between both tumors. At first glance the tumor of the esophagus seems to be simply a recurrence, which became manifest three years after the removal of the retroesophagus tumor and which, according to de Quervain,[1] is frequently observed in fibrosarcoma of the neck; however, the different location of both tumors requires a more exact analysis. According to Ewing,[3] recurrent tumors in the great majority of cases arise from cells or portions of the tumor which have escaped removal. This recurrence is usually prompt, multiple, in the line of incision or near by and of the same type as the original growth, or more malignant. It is apparent that the tumor of the esophagus in the present case meets only with the

requirement concerning the structure of the tumor. Furthermore, the retroesophageal tumor was easily removed from the esophagus by dull dissection with the finger, so that it is hardly conceivable that portions of the tumor escaped removal; therefore, the tumor of the esophagus in the present case was not a true recurrence.

As a second source of recurrence, it was suggested that tumors exert a pervasive formative influence, leaving the surrounding tissue in a state of excitation and with a momentum toward neoplastic growth, so that after the removal of the growth, the adjacent tissues give origin to a new tumor (Ewing[3]). It would be possible to classify the tumor of the esophagus in the present case as recurrence of that type. The analysis of the case history does not conform with that concept, as immediately after the operation a complete laryngeal recurrent paralysis on the left side was observed. Whether or not the paralysis was present prior to the operation cannot be stated, as it was not possible to see the larynx prior to the operation by means of indirect laryngoscopy. It was assumed, therefore, that the paralysis was due to the surgical procedure. In that case an improvement of the paralysis was to be expected, inasmuch as the nerve could be only traumatized by the dull dissection but not cut through. Further observation, however, proved the paralysis to be permanent. This leads to the conclusion that the paralysis was not due to the surgical procedure but to the sarcoma of the esophagus; *viz.*, that the sarcoma of the esophagus was present when the retroesophagus tumor was removed. In other words, the sarcoma of the esophagus did not make its appearance after or due to the removal of the retroesophageal tumor. It is more probable that it was present prior to the removal of the latter.

A third source of recurrence is found in the multiple origin of many tumors (Ewing[3]). In such instance the sarcoma of the esophagus cannot be considered as true recurrence but as a new tumor becoming manifest after the operation. According to Ewing,[3] these new tumors do not appear in the scar but they develop usually after an interval, reproduce the original growth without great change in malignancy and are often solitary. Unquestionably, the tumor of the esophagus in the presented case fulfills almost all of these requirements.

SUMMARY.

A case of two spindle-cell sarcomas of the neck was described in a luetic individual. The first tumor had an extra-esophageal location between the esophagus and vertebra, and in three years attained the size of a grapefruit (weight 345 gm.), did not produce metastases or a recurrence and was removed by surgery. The second tumor had an intra-esophageal location within the anterior wall of the upper third of the esophagus, attained a considerable size in three years, although less than the first tumor, and perforated into the lumen of the esophagus, but not into the trachea, leading to fatality due to hemorrhage and aspiration of blood into the lungs.

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307 North Michigan Boulevard.

PRINCIPLES OF DIAGNOSIS AND TREATMENT OF ALLERGY AS RELATED TO OTOLARYNGOLOGY.*

DR. FRENCH K. HANSEL, St. Louis.

The various manifestations of allergy present an important problem of diagnosis and treatment in all branches of medicine, for almost any structure in the body may be involved. There is definite evidence that the incidence of allergy among the general population is increasing. In recent years greater contact with industrial dusts, house and automobile dust, atmospheric molds and pollens has increased the incidence of respiratory allergy. Exposure to the massive atmospheric concentration of ragweed pollen, especially in the Central States area, is inducing more and more cases of hay fever. Cross-country motor travel and the custom of frequenting ragweed areas during the period of maximum pollination result in even greater massive exposure. After the onset of ragweed hay fever, other sensitivities, such as those to dusts and foods, are frequently added, consequently the patient develops other manifestations of the disease. Contact dermatitis has become more common because of greater contact with a variety of chemical substances especially in the industries. Sensitivity to various drugs, vaccines, serums and endocrine products is not uncommon.

The solution of the problem of diagnosis and treatment is dependent primarily upon a complete clinical history and physical and laboratory examination including diagnostic skin tests. From the clinical history it is important, first, to determine whether one or more manifestations of allergy are present. The most common manifestations to be considered are:

Group I: Perennial nasal and sinus allergy, allergy of the eye and ear, hay fever, allergic bronchitis and bronchial asthma.

Group II: Gastrointestinal allergy, allergy of the salivary glands, allergic headache, urticaria, angioneurotic edema and eczema.

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Group III: Serum disease.

Group IV: Contact dermatitis.

The general practitioner has the occasion to deal with all these various manifestations while the specialist may see only those which are concerned with his particular field. Not infrequently, the patient has more than one manifestation; but he usually consults someone about that manifestation which dominates the clinical picture. The otolaryngologist, therefore, is particularly concerned with those manifestations listed in Group I, although one or more of those in Group II may be associated. The internist has occasion to observe especially those patients in Group I with bronchitis and asthma and those in Group II. Although the pediatrician may contact patients in all groups, most of them fall in Groups I and II. The dermatologist is concerned with the skin allergies in Group II and contact dermatitis of Group IV. It is important that each specialist recognize those associated manifestations which are not primarily concerned in his specialty.

From the standpoint of the otolaryngologist it should also be emphasized that the ear may be involved by the allergic reaction. In the auditory canal and on the auricle eczema and contact dermatitis are not uncommon. Although allergic otitis media has been described, there is still some question as to whether or not this is possible since among those cases reported, eosinophiles in the discharge have not been noted. In some cases of nasal allergy we have noted involvement of the Eustachian tube giving rise to symptoms of tubal obstruction including deafness. The cochlea and labyrinth may also be involved. In certain cases of Ménière's disease allergy has been found to be the cause of symptoms. Auditory nerve deafness as a complication of serum disease has been reported by Cutter.[1] As a matter of fact, all the cranial nerves, individually or collectively, have been reported as paralyzed as a complication of serum disease.

It has recently come to our attention that allergy may affect the salivary glands. During the past three years five cases have come under our observation. In 1935, Pearson[2] reported 17 cases of allergy of the parotid gland and in 1936 two more cases were added.[3] Rowe reported one case in which the submaxillary gland was involved. Allergy of the parotid

is characterized by the appearance of unilateral or bilateral painless swellings. The swelling may subside upon massaging Stenson's duct, from which a thick mucus plug containing eosinophiles may be expressed. Pearson injected the ducts with lipiodol, then by means of the X-ray showed that there was a dilatation of the ducts. The swelling of the gland, therefore, is the result of obstruction of Stenson's duct rather than edema of the gland. Allergy of the parotid is usually associated with other manifestations and is usually caused by foods. Pearson reported two cases in which the swellings were associated with attacks of asthma. In children they not infrequently occur with attacks of abdominal pain or colic. In some instances secondary infection in the gland may complicate the allergic process.

In children as well as in adults allergy must be differentiated from recurring head colds. The tonsils and adenoids are not infrequently removed in children for the relief of frequent colds and bronchitis when the cause of the symptoms may be due to allergy rather than to infection.[4] In a group of 200 children who were routine candidates for removal of the tonsils and adenoids we[5] found an incidence of respiratory allergy of 13 per cent. Chronic cough in children often precedes asthma; therefore, allergy must always be considered as a possible cause.

The diagnosis of respiratory allergy can usually be established without difficulty upon the basis of the clinical history and the physical examination.[6,7] In dealing with the nose and paranasal sinuses, however, there should be an additional correlation of the cytologic examination of the secretions, the X-ray and bacteriologic findings. This plan of diagnosis should also be employed in the management of bronchial asthma. The cytologic examination of the nasal and bronchial secretions is important not only from the diagnostic standpoint but also from the viewpoint of determining the presence or absence of complicating infection. In uncomplicated cases the secretions show a pure eosinophilia. With secondary chronic infection there is usually a mixture of eosinophiles and neutrophiles. During an acute cold or in the presence of chronic suppuration the neutrophiles predominate the cytologic picture. If severe, the eosinophilia may completely disappear. This is particularly true during the

active stage of an acute cold. The eosinophilia returns with the subsidence of the infection. Cytologic examinations are important in determining whether an acute exacerbation of symptoms is caused by allergic reaction or by the presence of infection. They are also a guide, therefore, in the treatment of the immediate symptoms. Chronic allergic cough in the absence of definite nasal symptoms or asthma may be diagnosed only on a cytologic basis. From the standpoint of the X-ray examination, a diagnosis of acute or chronic sinusitis should not be made without the correlation of the cytologic findings.

Before adopting any definite plan of treatment of the allergic patient it is important primarily to determine which manifestations of the disease are present. In general it may be stated that the type or types of allergy present give some clew as to the cause of symptoms. For example, the respiratory allergies listed in Group I are in the majority of cases caused by inhalant substances such as pollens, atmospheric molds, house dust, occupational dust, cosmetics, animal emanations. Only in a small percentage of cases is food a factor if respiratory allergy only is present. Those manifestations listed in Group II are caused almost exclusively by ingested substances such as foods and sometimes drugs. The coexistence of Group I and Group II manifestations indicates that both inhalant and food factors play a part. Food is rarely the sole cause of respiratory allergy.

In the determination of the etiologic factors the information derived from skin tests should be correlated with the clinical history. In the case of the pollen allergies they are very reliable, but in the inhalant and food cases they may or may not be of value. Inhalant or respiratory allergy such as the house dust type, for example, may be present with negative skin reactions. In these cases dust therapy should be employed. Likewise in the food cases skin tests may be unreliable; therefore, some type of elimination diet must be employed to determine the causative factors.

In some instances the clinical history alone may reveal the inhalant and food factors, the avoidance or elimination of which may result in the satisfactory relief of symptoms.

In the management of respiratory allergy certain prophylactic measures should be instituted as a routine procedure.

Pillows and mattresses should be enclosed in dustproof encasings. The household should be kept as meticulously free of house dust as possible. The nonallergic type of cosmetics should be used. Household pets should be eliminated. A change of occupation or residence may be indicated in some instances. If the removal or avoidance of inhalant factors is not possible or is insufficient to give satisfactory relief, treatment by the injection of an extract of the offending substance is indicated. In some cases injection therapy must be used in conjunction with food avoidance.

Among the inhalant allergies, exclusive of hay fever, house dust is the most common cause.[8] Autogenous house dust or a well compounded stock dust extract may be employed. In those instances in which some specific dust is the factor, an extract of it should be used. In some cases equal parts of autogenous house dust or stock house dust and the specific dust must be combined to insure added protection or to produce satisfactory relief.

PREPARATION AND ADMINISTRATION OF DUST EXTRACTS AND OTHER ANTIGENS.

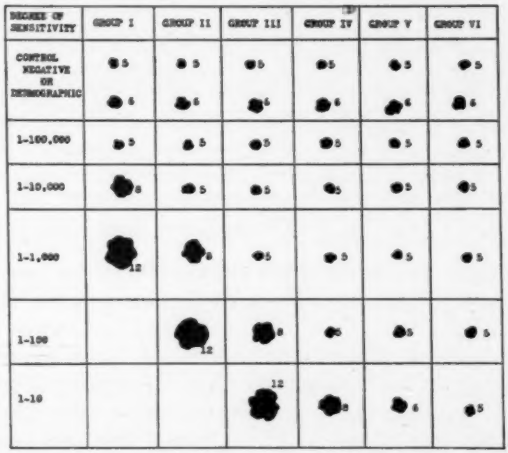
The following instructions for the dilution, titration of skin reactions and the administration of stock dust are also applicable for related antigens such as autogenous house or occupational dusts, animal danders, orris root, pollens or for foods. Concentrated stock dust or other antigens may be extracted with glycero-saline (50 per cent glycerine, 50 per cent buffered saline) or coca solution. All dilutions are made with coca solution (phenol 4 gm., sodium bicarbonate 2.75 gm. and sodium chloride 5 gm. per 1,000 cc. of distilled water). Extracts must be sterilized by Seitz or Berkefeld filtration.

The concentrated stock dust or other antigen may be diluted as follows: 1:10, 2 cc.; 1:100, 5 cc.; 1:1,000, 10 cc.; 1:10,000, 10 cc.; 1:100,000, 10 cc.; 1:1,000,000, 5 cc.; 1:10,000,000, 5 cc. It may be diluted in larger quantities if necessary. Fresh dilutions should be prepared every 30 days as there is a loss of potency upon dilution and standing. Solutions should be kept in a refrigerator if possible, otherwise they should be stored in a dark, cool place.

The degree of skin sensitivity is determined by the intracutaneous method. About 0.02 cc. of extract is injected, mak-

ing a wheal about 3 mm. in diameter. For this purpose we use a 1 cc. graduated tuberculin syringe, 26-gauge needle, $\frac{5}{8}$-inch long. Tests should be made first with the 1:100,000, 1:10,000 and 1:1,000 dilutions and compared with a control of coca solution (see Chart 1). In very sensitive patients, such as those in Group I, additional tests should not be applied. In moderately sensitive patients, such as those in Group II, the 1:100 dilution may be added. In less sensitive patients, such as those in Group III, the 1:100 and 1:10 dilutions may be added.

CHART 1. TITRATION OF DUST SENSITIVITY.



In patients with good reacting skins the titration reactions are a reliable index of sensitivity, but in poorly reacting skins the reactions are less reliable. In dermographic skins there may be a slightly positive reaction to the control, in which instance all dilutions will show some reaction. If, for example, in Group I, a dermographic reaction occurs to the control and the 1:100,000 shows the same size wheal, then the latter is also a dermographic reaction and not a positive reaction. On the other hand, the patient may be more sensitive clinically than the skin reaction indicates.

Most satisfactory results are dependent upon optimum dosage. Poor results are usually due to starting with a dilution

which is too strong or overstepping the tolerance with a dosage which is too high; therefore, the increase may be too rapid or optimum dosage may be overstepped. According to the schedule in Table I, it should be noted that, for example,

TABLE I. TREATMENT BY SUBCUTANEOUS INJECTIONS — STARTING WITH FIVE- TO SEVEN-DAY INTERVALS.

1. 0.10 cc. 1:100,000	18. 0.45 cc. 1:10,000
2. 0.15 cc. 1:100,000	19. 0.50 cc. 1:10,000
3. 0.20 cc. 1:100,000	20. 0.05 cc. 1:1,000
4. 0.25 cc. 1:100,000	21. 0.10 cc. 1:1,000
5. 0.30 cc. 1:100,000	22. 0.15 cc. 1:1,000
6. 0.35 cc. 1:100,000	23. 0.20 cc. 1:1,000
7. 0.40 cc. 1:100,000	24. 0.25 cc. 1:1,000
8. 0.45 cc. 1:100,000	25. 0.30 cc. 1:1,000
9. 0.50 cc. 1:100,000	26. 0.35 cc. 1:1,000
10. 0.05 cc. 1:10,000	27. 0.40 cc. 1:1,000
11. 0.10 cc. 1:10,000	28. 0.45 cc. 1:1,000
12. 0.15 cc. 1:10,000	29. 0.50 cc. 1:1,000
13. 0.20 cc. 1:10,000	30. 0.05 cc. 1:100
14. 0.25 cc. 1:10,000	31. 0.10 cc. 1:100
15. 0.30 cc. 1:10,000	32. 0.15 cc. 1:100
16. 0.35 cc. 1:10,000	33. 0.20 cc. 1:100
17. 0.40 cc. 1:10,000	

After optimum dosage is reached, gradually decrease the intervals between injections to 10, 14, 21 days.

the thirteenth dose is 20 times stronger than the first. Maximum dosage is optimum dosage, or the one which gives satisfactory relief. When this point is reached, an increase in the intervals between treatments to 10 days, then 14 days and finally 20 to 30 days should be tried without increasing the dosage. If the intervals cannot be increased, then one or two higher doses may be given. The optimum dose may be higher, in other instances lower, than those tabulated.

Among those patients with marked nasal allergy or asthma, treatment should be started with 0.10 cc. of 1:100,000. The optimum dose may vary from 0.10 cc. of 1:10,000 to 0.10 to 0.20 cc. of 1:1,000. In extremely severe cases it may be advisable to start treatment with the 1:1,000,000 dilution. In cases of less than average severity the 1:10,000 dilution may be used first. It is more advisable to determine the dosage on the basis of the degree of symptoms rather than on the degree of skin reactivity noted in the titration. In small children between 2 and 6 years of age treatment should begin with the 1:10,000,000 dilution. In children from 6 to 15 years of age the 1:1,000,000 dilution should be used first.

If in any instance there is an exacerbation of symptoms after any individual injection, the dosage should be reduced about 10 times.

In some cases treatment may be necessary only during the fall and winter months; in others, it must be given perennially. After one or two years some patients do not require further treatment. It is always advisable to institute prophylactic measures as already suggested.

We have noted that among patients sensitive to house dust that in general the symptoms are definitely more marked during the fall and winter months. Those patients who still have marked symptoms during the spring and summer months may be sensitive to atmospheric molds. In these cases the extract should consist of half stock dust and half feed mill dust which contains all the important atmospheric molds such as alternaria, hormodendrum, helminthosporium, rusts and smuts. Furthermore, we have noted that some patients have nasal symptoms and asthma only during the summer months as the result of mold sensitivity. These patients may or may not be sensitive also to pollens.

The following case reports illustrate the plan of treatment employed and the results obtained:

Case 1: Mrs. A. Z., age 52, nasal allergy with pharyngeal irritation. Sneezing, nasal obstruction and discharge had been present for 10 years. The examination of the nose showed a pale, boggy mucosa. The secretions contained a moderate number of eosinophiles. The skin tests were all negative.

Stock dust therapy was administered as follows:
July 21, 1941: 0.10 cc. 1:10,000 No. 18 dust.
July 30, 1941: 0.15 cc. 1:10,000 No. 18 dust.
Aug. 6, 1941: Marked improvement; 0.20 cc. 1:10,000 No. 18 dust.
Aug. 13, 1941: 0.25 cc. 1:10,000 No. 18 dust with molds 1:100,000.
Aug. 22, 1941: Relief practically complete; 0.30 cc. 1:10,000.
Aug. 29, 1941: 0.35 cc. 1:10,000.
Sept. 5, 1941: 0.40 cc. 1:10,000.
Sept. 15, 1941: 0.45 cc. 1:10,000.
Oct. 16, 1941: 0.10 cc. 1:1,000.
Nov. 21, 1941: 0.10 cc. 1:1,000.
Dec. 3, 1941: 0.15 cc. 1:1,000.
Dec. 19, 1941: 0.20 cc. 1:1,000.

This case illustrates the response to low dosage in spite of negative skin reactions.

Case 2: Mrs. J. M. O., age 50, July 19, 1939. Chief complaints: sneezing, nasal obstruction and discharge, moderate cough and wheezing. Mild nasal symptoms had been present since childhood. Since June, 1934, the nasal symptoms had been very marked. Removal of nasal polyps and

ionization were performed in August, 1934. A perforation of cartilaginous septum followed the ionization. There was no relief of symptoms following these procedures. The examination of the nose showed a very pale, boggy mucosa with almost complete obstruction of both sides of the nose. Polyposis, grade 3, right; grade 2, left. Perforation of cartilaginous septum about 1 cm. in diameter. The nasal secretions showed a marked eosinophilia. The skin tests were all negative.

Stock dust therapy was instituted as follows:

July 19, 1939: 0.10 cc. 1:1,000 No. 14 stock dust.
July 24, 1939. Definite relief after first injection 0.20 cc. 1:1,000 No. 14.
July 28, 1939: Very marked relief of symptoms. Nose definitely improved; 0.10 cc. 1:1000 No. 14 with molds 1:10,000.
Aug. 2, 1939: 0.20 cc. 1:1,000 No. 14 with molds 1:10,000.
Aug. 7, 1939: 0.30 cc. 1:1,000 No. 14 with molds 1:10,000.
Aug. 14, 1939: 0.40 cc. 1:1,000 No. 14 with molds 1:10,000.
Aug. 23, 1939: Slight increase in symptoms; 0.20 cc. 1:1,000.
Sept. 1, 1939: No nasal polyps; nose open and free of symptoms; 0.20 cc. 1:1,000.
Sept. 11, 1939: 0.20 cc. 1:1,000.
Sept. 20, 1939: Slight return of symptoms; 0.10 cc. 1:1,000.
Sept. 20, 1939 to Dec. 18, 1941: Maintained on 0.10 cc. 1:1,000 solution.

Case 3: Mrs. J. T. J., age 30, Nov. 6, 1941. Very marked nasal allergy of three years' duration. Uses 250 Kleenex a day. Nasal mucosa moderately pale. Secretion thin, moderate eosinophilia. Skin reactions all negative.

Stock dust therapy:

Nov. 14, 1941: 0.10 cc. 1:10,000 No. 19 W.
Nov. 21, 1941: Marked improvement; 0.15 cc. 1:10,000.
Nov. 28, 1941: Practically complete relief; 0.20 cc. 1:10,000.
Dec. 5, 1941. 0.25 cc. 1:10,000.
Dec. 9, 1941: Slight return of symptoms; 0.10 cc. 1:100,000 No. 19 W.
Dec. 16, 1941: Free of symptoms; 0.15 cc. 1:100,000.
Dec. 23, 1941: 0.20 cc. 1:100,000.

Case 4: R. M., age 24, Aug. 16, 1941. Nasal allergy and bronchial asthma. Nasal allergy 10 years. Asthma since December, 1940. Occupational dust contact. Weight 140. Nasal mucosa pale and boggy. Moderate eosinophilia of nasal and bronchial secretion. Skin reactions positive to house and occupational dusts.

Weight 140 —

Aug. 18, 1941: 0.05 cc. 1:10,000 No. 18; 0.05 cc. 1:10,000 occupational dust.
Aug. 24, 1941: Worse after first injection; 0.10 cc. 1:100,000 No. 18—1:10,000 occupational dust.
Aug. 30, 1941: 0.10 cc. 1:100,000.
Sept. 5, 1941: 0.15 cc. 1:100,000.
Sept. 13, 1941: 0.20 cc. 1:100,000.
Sept. 19, 1941: 0.25 cc. 1:100,000.

Weight 147 —

Sept. 27, 1941: Occasional mild attack; 0.30 cc. 1:100,000 No. 18 W—1:10,000 occupational dust.
Oct. 5, 1941: 0.35 cc.
Oct. 11, 1941: 0.40 cc.
Oct. 17, 1941: 0.20 cc.; mild symptoms after last injection.
Oct. 24, 1941: 0.25 cc.
Oct. 31, 1941: 0.30 cc.

Nov. 9, 1941: 0.35 cc.
Nov. 19, 1941: 0.40 cc.
Dec. 1, 1941: 0.45 cc.

Weight 151—

Dec. 14, 1941: 0.50 cc.

Patient has been asthma-free to date.

Case 5: Miss O. L., age 19, Aug. 15, 1941. Marked nasal allergy of 10 years' duration. Loss of sense of smell. Very pale, boggy mucosa with polyposis, grade 1. Marked secretion eosinophilia. Many +1 skin reactions to foods and inhalants. No food manifestations. Positive skin reaction to 1:100 stock dust.

Stock dust treatment:

Aug. 20, 1941: 0.05 cc. 1:10,000 No. 18.
Aug. 25, 1941: 0.10 cc.
Aug. 29, 1941: 0.15 cc.
Sept. 5, 1941: 0.20 cc.
Sept. 12, 1941: 0.25 cc.
Sept. 23, 1941: 0.30 cc.
Sept. 29, 1941: 0.35 cc.
Oct. 6, 1941: 0.40 cc.
Oct. 13, 1941: 0.45 cc.
Oct. 24, 1941: 0.50 cc.
Nov. 3, 1941: 0.10 cc. 1:1,000 No. 18.
Nov. 17, 1941: 0.15 cc.
Dec. 12, 1941: 0.10 cc.

Gradual improvement with complete relief and a marked change in the nasal mucosa toward normal color. Nasal obstruction relieved. Return of sense of smell for first time since early childhood.

THE DIAGNOSIS AND TREATMENT OF HAY FEVER.

In the diagnosis and treatment of hay fever the following factors must be taken into consideration:

1. The specific pollens which appear in the atmosphere in a given locality.

2. The pollens to which the patient is sensitive.

3. The degree of sensitivity.

4. The establishment of an optimum effective dosage.

5. The consideration of nonpollen sensitivities and the existence of other manifestations of allergy.

Among the many varieties of trees, grasses and weeds, only a comparatively small number produce enough atmospheric pollen to be of significance. The evaluation of the plants which cause hay fever is based upon their relative abundance and distribution and upon the amount of pollen which appears

in the air. Field surveys, therefore, must be correlated with atmospheric counts.[9] The technique of making these counts is as follows: *1.* A microscopic slide is coated with glycerine jelly and exposed for 24 hours; *2.* basic fuchsin may be added to the jelly to give it the color of red ink — two to three drops of saturated aqueous solution to one ounce. This stains the pollen grains a purplish-red color and makes identification more easy, as mold spores and other materials do not take the stain; *3.* the number of pollen grains appearing on an area of 1.8 sq. cm. is considered the atmospheric count. This also represents the number of grains per cubic yard of air in 24 hours. In general, the atmospheric count must be 25 or over to cause symptoms.

In the city of New York and vicinity the hay fever season begins about the first of April with the pollination of the trees. The grasses begin in late May, the goosefoots in July and the ragweeds in August. The following schedule, according to Wodehouse,[10] shows the common causative wind-borne pollen factors in hay fever and their approximate dates of pollination:

Trees —

Cedar (Juniperus)	March 15 to May 15
Alder (Alnus)	April 1 to April 25
Elm (Ulmus)	April 1 to May 1
Maple (Acer)	April 1 to May 10
Willow (Salix)	April 1 to May 30
Poplar (Populus)	April 15 to May 15
Birch (Betula)	May 1 to June 1
Sycamore (Platanus)	May 5 to June 1
Ash (Fraxinus)	May 10 to May 25
Oak (Quercus)	May 10 to May 30
Walnut (Juglans)	May 15 to June 15
Hickory (Carya)	May 15 to June 15

Grasses —

Sweet vernalgrass (Anthoxanthum odoratum)	May 1 to June 25
June grass (Poa pratensis)	May 25 to July 1
Orchard grass (Dactylis glomerata)	June 1 to July 20
Redtop (Agrostis alba)	June 15 to Aug. 1
Timothy (Phleum pratense)	June 25 to Oct. 1

Weeds —

English plantain (Plantago lanceolata)	May 10 to Sept. 15
Goosefoot (Chenopodium album)	July 1 to Oct. 1
Pigweed (Amaranthus retroflexus)	July 15 to Oct. 1
Sunflower (Helianthus annus)	Aug. 1 to frost
Short ragweed (Ambrosia elatiro)	Aug. 10 to October
Giant ragweed (Ambrosia trifida)	Aug. 10 to October
Goldenrod (Solidago speciosa)	Aug. 15 to frost

Mugwort (Artemisia absinthium)	Aug. 15 to frost
Cocklebur (Xanthium)	Aug. 20 to frost

In the diagnosis of hay fever the skin tests to pollen extracts are very reliable. The occurrence of hay fever in the absence of positive skin reactions is very rare. On the other hand, false positive reactions may occasionally be noted in the absence of hay fever. In the final diagnosis the skin reactions should be correlated with the time of appearance of the symptoms, the atmospheric counts and field surveys. The small group of tree, grass and weed pollens enumerated above are sufficient for skin tests. A larger number is not necessary. For diagnostic purposes we routinely use the cut or scratch method.

For the purpose of determining the degree of skin sensitivity and for treatment the pollen extracts should be made into individual or combined solutions of various dilutions from 1:100 to 1:10,000,000. The extracts may be used individually or combined, according to which ones are a factor. For the grass hay fever which begins about May 15, June and orchard grass should be used in equal parts. Plantain or dock may be used individually or combined with the grasses. After June 15, timothy and red top should be used for grass hay fever which occurs at this time. For the ragweeds and the goosefoots, combined extracts of these two families may be used as individual or as combined mixtures according to whether the patient is sensitive to ragweeds or goosefoots, or both. If necessary, grass extracts of those which pollinate at this time may be added.

With patients who do not receive satisfactory results from preseasonal subcutaneous injection therapy after the season begins, the intracutaneous method may be used and the subcutaneous injections discontinued. Intracutaneous treatment may also be found to be most satisfactory with patients who had had incomplete preseasonal therapy. Whenever possible the coseasonal type of therapy should be preceded by a series of about 10 injections, either intracutaneously or subcutaneously, before the onset of the season.

During the past six years we have employed the coseasonal method of treatment in practically all the tree and grass cases and in about one-half of the ragweed cases with most satis-

factory results.[11] Most of the patients had ragweed hay fever; many of these did not report for treatment until after the season had started. Some patients were treated by the intracutaneous method for six consecutive years.

Before the initial dosage is given, the skin sensitivity is determined by intracutaneous tests with 0.01-0.02 cc. of the various dilutions ranging from 1:10,000,000 to 1:1,000 (see Chart 2). Occasionally the 1:100,000,000 or the 1:100 dilu-

CHART 2. TREATMENT OF HAY FEVER. TREES, GRASSES, WEEDS. TITRATION OF SENSITIVITY.

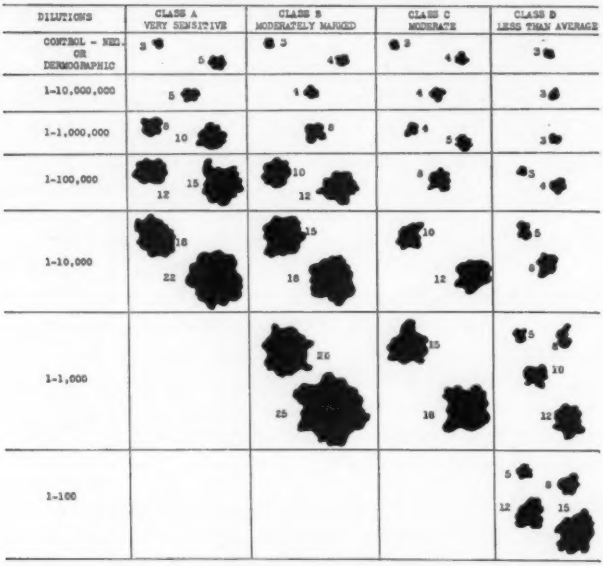


Chart 2. Diameter of wheals in millimeters. Always test first with 1-10,000,000, 1-1,000,000 and 1-100,000 dilutions; then add 1-100,000 and 1-1,000 according to sensitivity.

tions are necessary. Initial testing is always made with the 1:10,000,000, 1:1,000,000 and the 1:100,000 dilutions using coca solution as a control. If a positive whealing reaction as large as 15 mm. in diameter is noted to the 1:100,000 dilution, no further tests are made at the initial sitting. Within one or two days, treatment should be started with 0.02 cc. of the 1:100,000 dilution, which usually produces a wheal about

15 to 18 mm. in diameter. Subsequent doses should be administered every two to five days or longer, according to the degree of relief obtained. The amount of the injections should be increased by 0.01 cc. The size of the wheal increases with each subsequent dosage by about 1 to 2 mm. The average size wheal which produces satisfactory relief varies from 18 to 22 mm. in diameter. Occasionaly it may be necessary to increase it from 25 to 30 mm. in diameter. In order to reach optimum dosage, as much as 0.05 cc. of the 1:10,000 dilution may be necessary in cases of moderately marked sensitivity. After a few treatments at two- to three-day intervals it is often possible to increase the intervals from five to seven days in ragweed cases. In the tree and grass cases longer intervals are often possible because of the marked variations in pollen concentration. In some of the grass cases, intervals of two weeks or longer are possible.

With each subsequent treatment there is a tendency to reduction of the skin reaction in proportion to the dosage injected.

By this method of treatment the size of the skin reaction, the degree of relief and the intervals between injections indicate the optimum dosage.

In the cases of moderately marked sensitivity a wheal about 15 mm. in diameter is the rule with 0.01 cc. of the 1:10,000 solution. The 1:1,000 should not be applied at the first sitting. Treatment should be continued with increasing doses of 0.02, 0.03, 0.04 and 0.05 cc., and then with 0.01, 0.02, 0.03, 0.04 and 0.05 cc. of the 1:1,000 dilution according to the plan outlined above. Satisfactory response is usually noted to wheals varying from 18 to 22 mm. in diameter.

In patients with moderate sensitivity, a wheal about 15 mm. in diameter is usually obtained with the 1:1,000 dilution. Subsequent injections should be increased by 0.01 cc. until the proper size wheal is obtained. With the 1:1,000 dilution the amount of the injection may be increased up to 0.05 or 0.07 cc. — higher if necessary.

In the patients with less than average sensitivity, the 1:1,000 dilution may be used throughout in doses up to 0.10 cc. It is rarely necessary to use the 1:100 dilution.

In giving subcutaneous injections by the preseasonal plan, small doses at five- to seven-day intervals have proved to be most satisfactory and free of complicating reactions. By this method an optimum dosage instead of a top dosage is desired. The original doses should start with 0.10 cc. of the 1:10,000,-000 or 1:1,000,000, according to the degree of skin reactivity, for Classes A and B, and increased by 0.05 cc. with each subsequent injection. Top doses should be about 0.10 cc. to 0.30 cc. of 1:100,000 in very sensitive patients (Class A), 0.10 to 0.30 cc. of 1:10,000 in the less sensitive (Class B), and 0.10 to 0.30 cc. of 1:1,000 in the least sensitive (Classes C and D). It is necessary to continue these injections every two to five days during the season, according to the symptoms and atmospheric pollen concentration.

For the most satisfactory results in hay fever treatment, complete skin testing should be performed and particular attention must be directed to the control of complicating sensitivities or manifestations such as those resulting from dusts, molds and foods.

SUMMARY.

1. The fundamental principles of diagnosis and treatment of the allergic diseases are based upon the analysis of a complete detailed clinical history, physical examination, skin tests and whatever additional laboratory procedures may be indicated.

2. Although some one manifestation usually dominates the clinical picture, the existence of others should be recognized when present.

3. In nasal, sinus and bronchial cases the following factors must be carefully evaluated: *1.* the symptoms; *2.* the gross pathologic changes; *3.* the cytology of the secretions; *4.* X-ray examination; *5.* the bacteriologic findings; and *6.* histopathologic findings. Acute, subacute and chronic complicating infections should be considered.

4. The removal or avoidance of the etiologic factors is important in treatment. Prophylactic measures should be instituted routinely.

5. Injection therapy for dust, mold and pollen sensitivities is most satisfactory following the plan of establishing a small optimum dosage.

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634 North Grand Boulevard.

AMERICAN BOARD OF OTOLARYNGOLOGY.

The next examination of the American Board of Otolaryngology will be held in New York at the Waldorf-Astoria Hotel and the New York Eye and Ear Infirmary on June 3-4-5, 1943. Direct all inquiries to Dr. D. M. Lierle, Secretary-Treasurer, University Hospital, Iowa City, Iowa.

PREVENTION OF DEFORMITIES IN CORRECTIVE RHINOPLASTY.

DR. MORTON I. BERSON, New York.

Plastic surgery, especially rhinoplasty, is unique in the realm of surgery in that the patient is the primary judge of the results obtained. The slightest defect, due either to a surgical or esthetic fault, causes great disappointment to the patient and in many instances may even affect him physically or mentally. To eliminate the possibility of defects, a thorough knowledge of surgical corrective procedure and a measure of artistic ability must be at the surgeon's command.

In this article, the author, confining himself to the most common type of deformed nose, namely, the congenital hypertrophied nose with hump, broad bridge, broad tip and excessive length, will endeavor to show: *1.* The possible deformities resulting from imperfect rhinoplastic technique; *2.* the proper technique to be employed in order to eliminate failures.

1. Undermining.

Improper undermining of the periosteum may cause:

a. Irregular lumping of the periosteum.

b. Periostitis.

c. Prolonged edema.

d. Irregular skin contractions.

The separation must be accomplished by use of the periosteum elevator rather than with scissors or other sharp instruments, in order to make the separation complete and to avoid tearing. It is important to feel the grind of the instrument against the bone to ascertain that it is under the periosteum. To avoid irregular lumping, severed shreds of tissue beneath the area of undermining must be removed. If this undermining is accomplished properly, the periosteum will contract smoothly and evenly over the bone.

2. Removal of Hump or Excess Height.

A. Bony section (upper part of nose).

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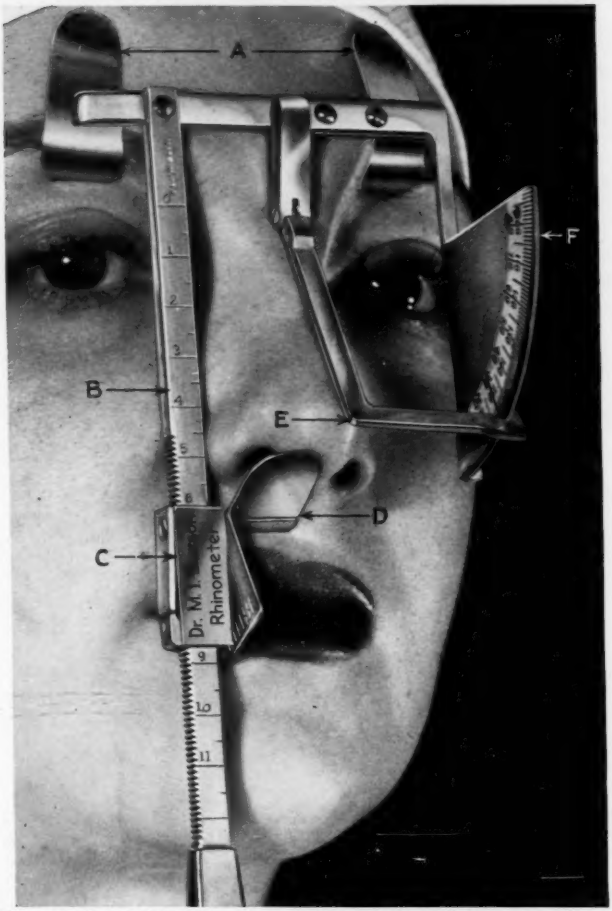

Fig. 1. Author's rhinometer. To obtain esthetic results in rhinoplasty, the author has devised a rhinometer. It is useful in the diagnosis of nasal deformities and is valuable in the progressive checking of the results during nasal operations. The instrument consists of the following parts:

(A) **Two rests** which are fitted under the superior orbital crests of each eye. These points are fixed and from them accurate measurements can be made.

(B) **A vertical column with a numerical scale** designed to carry a sliding platform.

(C) **A cross-member carriage,** designed to slide the platform to various levels along the column. Then, according to the graduated scale, is measured the difference between actual length (from nasion to apex) and the desired length.

(D) **The platform** fits under the columella of the nose and is tilted to the desired esthetic angle.

(E) **The lever on the calibrated arc** (F) is adjusted to the normal profile angle and the amount of excess height to be removed is determined.

By the use of the rhinometer, the following absolute measurements (as shown in the above illustration) can invariably be attained.

Profile angle is 30 degrees. Length of nose is one-third of facial length. Tip tilt angle is 95 degrees; this varies slightly according to the relationship with the lower third of the face.

The following errors should be avoided:

1. Removal of too much or too little dorsum.

2. Asymmetrical removal of bone, leaving irregularities of the bony bridge.

3. Failure to narrow nasal bony arch after hump removal, causing a wide, flat appearance.

4. Failure to correct the spring of the septum, causing a cartilage ridge in mid-dorsal section.

Fig. 2. Saw introduced through intercartilaginous incision. The bone, septal cartilage and perpendicular plate of the ethmoid cut through completely on each side.

To reduce the hump or height of the nose to the normal esthetic angle, the author's rhinometer (see Fig. 1) is placed over the nose with the lever adjusted to the lateral side, and a line is marked off with a dye at the 30° angle registered on the calibrated arc. A similarly measured line is also drawn on the other side of the nose. A section of hump is then removed according to preoperative markings (see Fig. 2). The saw is preferred to chisels and rasps, as by its use a more accurate amount can be removed more regularly. In attempting to remove the hump with rasps, a considerable amount of instrumentation is necessary, which produces an

added amount of injury, and spicules of bone may be left. Upon removing the section of hump (see Fig. 3) a definite spreading of the dorsum of the nose will occur, and the septum can be felt medially with the margins of the nasal bones on either side. This will insure that the nasal bones have been definitely cut across.

In deflected septums, especially when the perpendicular plate of the ethmoid is deviated, the dorsal margin of the septum will force itself through the fractured nasal bones and produce a definite sharp ridge in the mid-dorsal region months after the operation. To eliminate this, whenever the

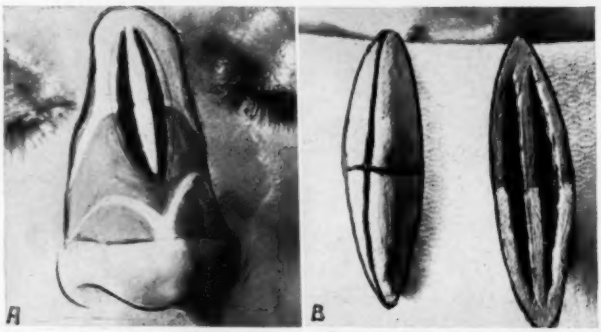

Fig. 3. (A) Dorsum after removal of hump, exposing free end of septum and margins of nasal bones on either side. (B) Hump after removal, showing outer and inner aspects of bone and cartilage.

septum has a definite deviation, a submucous resection must be done. It is, however, of great importance to leave a sufficient amount of dorsal and inferior part of the cartilage in the septum to give support to the dorsum of the nose after the hump has been removed, to avoid the possibility of saddle nose (see Fig. 4) and the dropping of the lower third of the nose.

B. Cartilaginous section (lower part of nose).

More than 75 per cent of all rhinoplastic defects occur in the lower third of the nose. The following factors produce these deformities:

1. Trauma of the soft tissues of the lower third, which

will cause the formation of an excessive amount of connective tissue and delay healing.

2. Failure to remove the proper amount of dorsum of septal cartilage.

3. Failure to remove foreign bodies.

4. Devitalized tissue produced by thrombosis of local blood vessels, which will act as a foreign body.

5. Dead spaces, which, though uninfected, will increase the volume of the wound and in that way lengthen the time required for complete healing.

Fig. 4. Saddle nose caused by resection of septum leaving no support over dorsum of nose. Corrected by removal of a section of nasal bone over the dorsum and transplantation of a strip of costal cartilage over the entire dorsum from glabella to tip.

6. Accumulation of exudate under tension such as hematomata will increase the volume of the wound and, by pressure, decrease the local blood supply and delay healing, even in the absence of infection.

7. Improper narrowing of the tip.

Elevation of the tip of the nose reveals the upper lateral cartilages attached to the septum medially. These cartilages are separated from their attachments to the septum on both sides and thus the dorsum of the lower third of the septum is freed. The cartilaginous dorsum, being exposed, will show a decided elevation since, in removing the hump the saw cuts through the bony structure but not through the mucous membrane and cartilage of the lower third.

To overcome this elevation, the cartilage of the dorsal septum must be excised to a lower level than the corrected bony structure, producing a slight concavity. Because of hyperplasia of sebaceous glands existing in the lower third of the nose, the healing skin will thicken and fill this concavity. Failure to remove that protruding section of cartilage and produce a concavity will cause the oval elevation (see Fig. 5), or parrotlike defect of the lower third of the nose. In cases where there are excess sebaceous glands, increased widening and thickening will result. X-ray or radium therapy, however, will reduce the hyperplasia of the sebaceous glands in these cases, lessening the edema and softening the scar tissue.

Fig. 5. Oval elevation of lower third of nose due to failure to remove sufficient dorsal septal cartilage and lower lateral cartilage. Corrected by reduction of this curve and additional narrowing, producing a finer esthetic appearance.

3. Narrowing the Tip.

Deformities resulting from improper narrowing of the tip:

a. Narrowing too much, producing a pinched-in tip.

b. Narrowing too little, leaving a broad tip.

c. Asymmetrical contractions of the tip and alae caused by unequal removal of cartilage.

The amount of cartilage to be removed is ascertained from the size of the tip. All supporting cartilage which produces

the abnormal width must be removed. Any small section of cartilage remaining in the tip will prohibit the contraction of the overlying tissues and cause indentations. The mucous membrane *must* be left intact, so that it will reattach itself to the underlying skin, eliminating the retraction of the lateral nasi which produces a pinched-in effect. When there is an indentation in the tip due to separation of the crus alae, the cartilage is removed if the tip is too broad; in a narrow tip, the crus alae are sutured together. Extreme care must be taken not to injure the skin in undermining, since it will leave a conspicuous scar.

Fig. 6. Dorsum of bridge remaining wide and flat after removal of hump and failure to narrow lateral walls of the nasal bones. Corrected by separating nasal bones in midline and completely fracturing them at nasomaxillary junction, then repositioning to the proper width.

4. Narrowing the Bridge.

Deformities produced by improper narrowing of the bridge:

a. Visible steplike projections on either side of the base of the nose.

b. Asymmetry of the sides.

c. Nasal obstruction due to failure to correct deviated septum before narrowing bridge.

d. Dorsum of bridge remaining wide and flat from failure to produce complete fracture of the lateral walls of the nasal bones (see Fig. 6).

After separating the periosteum (see Fig. 7), the saw is placed as in Fig. 8, in a straight line, equidistant on both sides. The saw is preferred to chisels because with its use a straight line is obtained more easily, and less trauma and

edema are caused. In most cases, after this sawing, the nasal bones can be refracted to the midline (see Fig. 9) by pressure with the thumbs. If the nasal bones do not fracture by pressure, a chisel 1 mm. wide is inserted through the eyebrow and passed under the skin to the frontonasal suture. Then, with the aid of a hammer, the nasal bone is fractured. This method will cause no visible scar and no irregularities of the nasofrontal junction. Should there be a slight elevation of

Fig. 7. Periosteum elevator inserted through lateral incision at nasal pyriformis. Skin and periosteum separated from lateral side of nose slightly anterior to the inner canthus as high as the glabella, to the dorsum of the nose and laterally over the nasofacial angle.

the maxillary bone, it can be reduced by the use of a chisel or rasp. To ascertain that the nasal bones are completely fractured at the nasomaxillary and nasofrontal areas, the bony bridge must be freely movable, and the opening over the dorsum of the nose should be closed by the uniting of the repositioned nasal bones.

5. Shortening the Nose.

Deformities produced by improper shortening:

a. Excessive shortening.

b. Insufficient shortening.

c. Displacement of the columella.

d. Conspicuous broadening of the nostrils.

The rhinometer will gauge the exact amount to be shortened (see Figs. 10 and 11), so that the final length of the nose will occupy one-third of the face and will be equal to the distance from the chin to the root of the nose and from the glabella to the hairline. Twenty per cent of the total should be added to the amount recorded on the rhinometer to

Fig. 8. Saw beneath periosteum, engaged in the nasomaxillary groove. The upper end of the saw is slightly anterior to the inner canthus, lower end is at the nasofacial groove.

allow for the average amount of postoperative drop. If the columella-lip junction forms a convexity or a shortening of the upper lip, caused by the protrusion of the anterior spine of the vomer, a triangular section of the nasal spine should be excised.

In shortening the nose or lessening the protrusion of the tip, the columella may displace itself to one side and cause a spread of the nostrils (see Fig. 12). This occurs through the

Fig. 9. Fractured bones shifted to midline, eliminating the dorsal broadness and re-establishing the line of arch.

Fig. 10. Forceps holding free margin of septum while with straight scissors, the measured excess amount of septal tissue is removed.

removal of a section of the posterior portion of the columella. To correct the displaced columella, it is detached at its lip

angle, the excess length is excised, and it is then resutured in the midline. To correct the spread of the nostrils, small crescentic sections are excised at the angle of attachment and the margins are sutured at the new width.

Fig. 11. Columella retracted to septal cartilage by two heavy silk sutures.

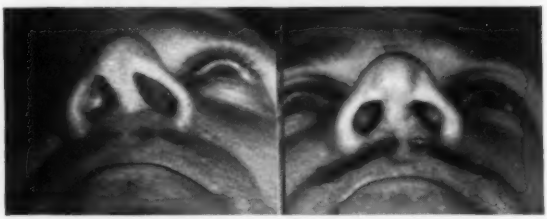

Fig. 12. Columella twisted and nostrils spread due to improper correction of tip protrusion. Corrected by repositioning columella and narrowing nostrils.

6. Distortion of the Nose Resulting from Overlapping of Tissue.

The entire field of the operation should be thoroughly checked for any irregularity, protrusion or loose shreds of

tissue. A protrusion of upper lateral cartilage will cause not only a visible flaring externally but also an irregular obstruction intranasally. This overlapping mucous membrane and cartilage must be excised and leveled down so that the loose margins approximate themselves.

7. Dressing.

The entire nasal passages are thoroughly cleaned and dried and gauze packing inserted to help approximate and hold the tissue in its new position. A supporting strip of adhesive is applied under the nose to approximate and hold the undersurface of the tip in the midline and provide support. Adhesive strips are then applied over the nose to hold in position and compress the tip. Under these strips of adhesive, gauze is applied to prevent the adhesive from adhering to the underlying skin and to allow the tissue to attach itself to the underlying structures. A stent mold is compressed firmly over the nose to immobilize the loosened bones in their new position and eliminate any dead space between the under- and overlying structures. This dressing should remain for five days to allow time for proper attachment.

Conclusions: The author, after a study of more than 500 cases, has found that in the absence of infection, failures in rhinoplasty can be completely eliminated by: *1.* Thorough study of the nose in each individual case; *2.* correct surgical procedure as outlined above; *3.* to obtain the best esthetic results, the author advocates the use, during the operation, of the rhinometer and a modeled nose on a mask, so that exact proportions in all dimensions can be more accurately reached.[1] It cannot be over-emphasized that each stage of a nasal plastic operation is equally important to the final result since failure in any one procedure may produce an artificial-appearing "plastic" nose or even a deformed nose.

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133 East 58th Street.

SINUSITIS AND OBESITY.*

DR. GEORGE KELEMEN, Boston.

Research into the correlation between sinus disease and nutritional factors has been conducted with intensity during the last two decades, and has concentrated on two points: *1.* The investigation was concerned chiefly with ingested food, especially vitamins; *2.* attention was focused on the tissues involved, as the mucous membrane and underlying osseous structures of the nasal and accessory cavities.

Dean[8] mentions Amy Daniels as the first to demonstrate, in 1918, the relationship between sinus infection and diets deficient in fat soluble vitamin A. This influence was believed to be so strong that even the superseding of surgery by purely dietetic treatment was discussed. Stucky[29] followed, in 1925, by offering diets deficient in vitamins as a factor in producing otolaryngological diseases. Barlow,[2] in 1928, studied otolaryngological manifestations in connection with vitamin deficiency, and Dutcher[9] discussed this question in 1929. Dean,[8] in 1929, stated that in the treatment of sinus infections a diet rich in vitamin A together with an increase in vitamin B is essential; but as early as this he already checked the initial overenthusiasm and advocated establishment of adequate drainage wherever there arose a question as to the amount of good that would be secured by administration of vitamins. He stated that the vitamins could not take the place of either the proper laryngological procedures or the observance of the usual hygienic regulations. Shurly[27] took up the question in 1928, then studied it in collaboration with Turner in 1929 and 1930. He emphasized a genuine practical application of dietetics in otolaryngology together with surgery. According to his conception, the demand of the human economy for iron, phosphorus, calcium, iodine and vitamins is greater in otolaryngological pathology than in other deficiencies of the body. In 1930, Lewis[16] advocated a dietary regimen in treatment of sinus conditions which consisted of a balance of protein, fat and carbohydrates, with

*From the Department of Otolaryngology, Tufts College Medical School. Aided by a grant from the Charlton Fund.

adequate intake of fluid. Fenton,[11] in 1930, emphasized that metabolic disturbances, mainly those of vitamin A deficiency, are among the most important factors in the etiology of sinus diseases. He expressed the hope that further studies in dietetics and nutrition would bid fair to solve many of the difficulties involved in these problems. Smyth,[28] in 1931, was able to review a series of authors who agreed that in sinusitis of children, proper diet was of the utmost importance. Cody[6] claimed, in 1931, that vitamin A was necessary for the maintenance of nutrition of the nasal, aural and tracheal mucosa; B deficiency manifested itself in a postnasal mucous discharge; metaplastic and inflammatory changes were influenced by a normal diet, so nutrition has to be improved in acute and chronic infections of these membranes. Sweet and K'ang,[30] in 1935, found that in vitamin A deficiency epithelial metaplasia similar to that noted in the eye occurred in the mucous membranes of the nose, accessory sinuses, trachea and bronchi. Jones,[12] in 1940, surveyed the rôle of vitamins in relation to the eye, ear, nose and throat. In the same year Selfridge[25] stated the fact that many conditions of the ear, nose and throat, among them sinus infections, allergic or non-allergic, are related to dietary errors: "germs are opportunists and must have a soil prepared." He gives Stucky[29] and Shurly[27] the honor of being the real pioneers in emphasizing that improper food intake is responsible for many of the troubles in the ear, nose and throat. Covell,[7] in 1940, described pathological changes in the peripheral auditory mechanism due to avitaminosis. Roberts,[24] in 1942, found that many otolaryngological complaints are practically always due to a nutritional disturbance.

This brief survey of some of the publications on the related question shows the trend of the discussion in the past few decades. All the authors seem to be in accord as to the influence of the general nutritional state on sinus conditions. In attempting to arrive at a better understanding of the nutritional factor as a whole, the aim has been to do so by evaluation of the particular food constituents.

The correlation between diseases of the upper airways and diet is discussed in some of the textbooks of otolaryngology. Lederer[14] states that overeating and an improper dietary regime render the body susceptible to upper respiratory dis-

eases. Logan Turner[31] finds it obvious that with an increase in knowledge of the part played both by diet and by allergy the methods of treatment of the air sinuses will require revision, more particularly, as far as prophylactic measures against infections are concerned.

One of the few attempts to carry dietary experience into practice was made by Moose.[18] Of his 300 analyzed patients, 73 per cent needed guidance in the choice of food. Among his cases, a woman, 35 years of age, was healed, by dietary changes, of sinus disorders which had persisted previously despite several operations. By the elimination of milk, a 58-year-old man was relieved from headaches and obstructed breathing: testing had found him sensitive to cow's milk. Moose[18] advocates the practice of taking a routine diet history in otolaryngology; the proper food may then be prescribed with other therapeutic measures. He considers the influence of dietary changes as follows: dietary change, chemical and physical constitutional change, change in the reaction to a disease, change in the course of the disease.

All this comprehensive work has not yet produced a true picture of integration, especially so because the overwhelming majority of the observations are based on animal experimentation with restricted applicability to human conditions.

Under those circumstances it may be of some value to use another approach — that is, study of the general condition as expressed by the body weight and the noting of the changes in the upper airways. Immediate opportunity was given by the following observation: At the Boston Dispensary many of our patients with sinus complaints, mostly of long standing, presented themselves with an obvious overweight. It seemed to be of interest to follow this coincidence and try to find a correlation between the two disorders.

The first step toward a general orientation was to take the *body weight* of 100 successive adult sinus patients, regardless of sex. As the evaluation of growth factors and other factors in connection with problems in children and adolescents, *e.g.*, the height-weight relation in these age groups, would have caused undesirable complications, patients under 20 years of age were excluded. From this age upward the series represents an unbroken line of patients as they presented them-

selves through November and December, 1941, and January, February and March, 1942. Only cases with underlying neoplasms were disregarded. With 16 males and 84 females, the ages of the patients varied between 20 and 75 years, in the following distribution:

TABLE 1.

Age	Males	Females
20 — 30	1	8
31 — 40	5	20
41 — 50	7	28
51 — 60	2	14
61 — 70	11	11
71 — 75	—	3

The average *height* of the males was 5.53 feet and that of the females 5.0 feet. These figures may be compared with average standards. The Mobilization Regulations call for men of a height between 5′4″ and 6′6″. The Department of Agriculture gives 5′3″ as average measurement of the American woman.

The weight was taken without shoes and overcoat, and in addition men were weighed without coat or sweater. From the figures obtained, five pounds were deducted as the final correction. The calculation of the height-weight relation was carried out on the basis of the tabulae in M. Fishbein's Handbook of Therapy, 11 ed., 1937. The deviation from the norm was then computed in percentages. Only one of the 100 showed the exact weight which corresponded to his height according to the tables. The others were found deviating from the norm as follows:

TABLE 2.

	Overweight			Underweight		
	Number of Patients	%		Number of Patients	%	
		Range	Average		Range	Average
Males	11	1.3-21.6	9.6	5	1.7-16.4	7.8
Females	61	2.0-92.1	24.3	22	0.7-26.5	10.9

Five pounds either way is not generally considered as abnormal; in exaggeration even of this allowance the next table disregards every person with a deviation from the norm to 10 pounds or less.

TABLE 3.

	Overweight		Underweight	
	Number of Patients	Pound Average	Number of Patients	Pound Average
Males	7	20.0	2	19.9
Females	49	34.4	11	20.9

In adding the figures for both sexes it was found that among the 100 patients, 69 had a deviation in either direction of more than 10 pounds; 56 persons had an overweight of a 32.6 pounds average, and 13 persons had an underweight of a 19.2 pounds average.

It is evident from this data that considerable overweight was far more frequent than considerable underweight, the ratio of the respective patients being 56:13. Besides this fact, the average of the overweight in poundage as contrasted with the average of the underweight in their deviation from the norm presents a ratio of 30.1:20.7.

To keep the consideration as far as possible between boundaries where safe deductions are possible, the patients with at least 20 per cent overweight were selected for a closer follow up. Concentrating on this group with re-examinations one could hope to get the most reliable data. In this group, there were only four men as against 34 women. The age of the males ranged between 46 and 70 years, with an average of 53.3; that of the females between 22 and 70 years, with an average of 43.0 years.

Only 13 patients were found to be underweight, with a deviation from the norm of more than 10 pounds; the average of this group was 19.2 pounds. Among the patients with underweight, there were five men and 22 women, with an average underweight of 7.8, respectively, 10.9 per cent.

The distribution concerning *nationality* as given by the patient is shown in the following table containing the percentage of the five most numerous groups. In this table, Column 1 is based on 100 patients seen at the Boston Dispensary in January, 1921; Column 2 is based on all the patients seen at the Food Clinic between Jan. 1 and Dec. 31, 1941; Column 3 is based on data concerning the 100 succes-

sive sinus patients; and Column 4 is based on data concerning 38 of the 100 patients of Column 3, presenting overweight of more than 20 per cent.

TABLE 4.

	1	2	3	4
American	27.8	35.7	23	13.2
Canadian	6.2	6.2	14	15.8
Irish	15.0	13.6	9	5.3
Italian	10.6	9.4	6	13.2
Jewish	16.2	15.4	33	26.3

The essentials of a few case histories are given as data characteristic of the whole group of 100.

Case 1: No. 161-700. White, male, 49 years, 5'5"; 185 pounds, overweight 21.6 per cent. Works as a tailor in a hot, steamy room. Under treatment at the Otolaryngological Department since 1928. Frequent colds, at certain periods almost constant, with headaches. Nasal obstruction with a deviated septum and hypertrophic turbinates. Discharge varying between watery and thick yellow in both nasal chambers. X-ray: pansinusitis, with polypoid degeneration of the mucous membrane in the ethmoids. Twice complicated with catarrhal otitis. Since the same year, 1928, has been under treatment at the Medical Department. The first diagnosis was: Obesity with gastric neurosis and constipation. BMR report, 1942: —1. No endocrine dysfunction. The Food Clinic tried without success to reduce the weight. Patient refused to stay on a strict diet, claiming it made him nervous.

Case 2: No. 179-762. White woman, 48 years, 5'1"; 167 pounds, overweight 22.7 per cent. A patient in the Otolaryngological Department since 1923, with catarrhal sinus condition. Headaches return regularly with frequent nasal blockage. Often suffers severe pains in the frontal and maxillary areas. She is in menopause; stilbestrol was tried. Since 1936, has had treatment for obesity. She allegedly tried very hard to lose weight, adhering to the prescribed diet, with only temporary success. BMR, 1942: +3. When not receiving constant local treatment, the nasal condition reappears within a short time with headaches and nonpurulent discharge.

Case 3: No. 202-197. This 43-year-old white woman presents a height of 4'11" and a weight of 160 pounds; overweight 28.1 per cent. Under treatment since 1933. The findings include purulent discharge with postnasal yellowish drip, obstructive septum, edematous turbinates, and recurrent sharp frontal headaches day and night. Highly emotional and in acute menopause since hysterectomy. Though very much pleased by any loss of weight with help of the dietary prescriptions of the Food Clinic, shows losses which are insignificant and temporary.

Case 4: No. 246-036. Thirty-eight-year-old white woman, 4'10" and 184 pounds, overweight 60.7 per cent. Under treatment since 1934 for recurrent nasal blockage, empyema of the antra, multiple polypi. Diagnosed as a psychoneurotic, the patient was found to be "very obese" by the Medical Department. Under thyroid treatment for four months, she lost only six pounds. Her excuse for not following the dietary prescriptions of the Food Clinic is that she had insufficient food money for special food. Hair-distribution of male type may represent ovarian failure (arrhenoblastoma).

Case 5: No. 262-018. White man of 48 years, 5'11" and 204 pounds, overweight 18.6 per cent. Treated since 1918 elsewhere for sinus disease;

has been a patient of the Otolaryngological Department since 1930. Headaches across the eyes, tenderness over all the four main sinuses, purulent nasal discharge. X-ray: both antra cloudy. Medical Department: excessive thirst, diabetes excluded. Feels that "reason he drinks so much water is that his nasal mucosa does not function properly." His weight always remains about the same.

Case 6: No. 311-840. Forty-four-year-old white woman, 5'3", 201 pounds, overweight 49.6 per cent. Under treatment since 1932. Constant nasal obstruction with positive X-ray findings, frontal and occipital headaches, starting generally in the afternoon. BMR: —14. No typical menopause symptoms. The Food Clinic finds that she has but little knowledge of food requirements; she emphasizes that her food money is not enough for adequate diet.

Case 7: No. 319-218. White woman, 45 years old; 4'7", 137 pounds, overweight 28 per cent. Chronic sinusitis with mucopus, hyperplastic turbinates, and nasal obstruction. The Food Clinic tries to keep her on an adequate diet without success. There is but very little loss in weight.

Case 8: No. 343-798. White woman, 42 years old, 4'11", 214 pounds, overweight 76.9 per cent. Under treatment since 1934 with nasal obstruction, purulent discharge and similar postnasal drip. The Food Clinic tries to instruct her about an adequate diet, but she makes very few attempts in this direction.

Case 9: No. 371-201. Thirty-six years old, white woman, 5', 244 pounds, overweight 92.1 per cent. Under treatment since 1939 with purulent rhinitis. Little relief from antral washes. Purulent discharge in nose and antra, postnasal drip, positive X-rays. BMR: —4. At the Food Clinic complains about scarce food money, but apparently follows diet. With her 92.1 per cent overweight she is the most excessive example of obesity among our 100 patients.

Case 10: No. 403-753. Thirty-one-year-old white woman, 5'5", 239 pounds, overweight 77 per cent. Complete nasal obstruction with mucopurulent discharge; under treatment for the last two years. Frontal headaches especially in the morning. Medical Department: diabetes, probably of pituitary type, otherwise endocrinology-study negative. Food Clinic: obesity believed to be exogenous. Not at all satisfied with prescribed diet; dislikes fruit and most vegetables: "only vegetable she can eat is tomato."

Case 11: No. 409-574. Thirty-six-year-old negro woman, 5', 197 pounds, overweight 61.5 per cent. Constant severe headaches across forehead, "heavy load on head." Nose blocked by edematous turbinates. In menopause. Food Clinic: she likes food too well. Does not follow the prescribed diet.

Case 12: No. 411-485. Forty-one-year-old white woman, 5'4", 179 pounds, overweight 29.8 per cent. Nasal blockage of allergic type with polypi, postnasal discharge, headaches, cloudy sinuses. BMR: —3. Medical Department: the upper respiratory condition is suggestive of possible correlation with ovarian dysfunction. Food Clinic: admits frequent overeating; never drinks water; has had the same overweight for the last 12 years.

The unusual high simultaneous occurrence of long standing nasal obstruction leading to sinus disease and obesity suggested an investigation regarding a deeper connection between the two conditions; the regularity of these findings seemed to occur too frequently for a simple coincidence.

Concerning the *sinus condition*, it must be emphasized that an underlying nasal occlusion was always found. Lack of

adequate drainage was thus a part of the pathology for the well known mechanism of retention of secretions with subsequent sinusitis. In three cases with positive Hinton tests, syphilis may have played a rôle and there were a few instances where dental origin was to be considered. No case was associated with acute infectious diseases, such as exanthemata or pneumonia. As usual, congested enlarged turbinates were of equal importance in producing the nasal obstruction along with septal deviations and spurs. The frequency of occurrence of the latter deformities was not surprising, but what was surprising was the low number of corrective septum operations, the more so since the complaints were long standing and operation obviously would have brought much relief. *Allergic state* played some part in this series of cases, but this was decidedly not of paramount importance. No case showed a distinct eosinophilia in nasal smears, according to Hansel's staining method.

It is remarkable that only relatively few severe infections were encountered which necessitated operative intervention. Only three cases demonstrated a tendency to spread beyond the boundary of the sinuses, as shown by temporary swelling of the soft tissues of the face over the sinuses. Relief could be brought about generally, at least temporarily, by the usual measures to improve ventilation; establishing satisfactory drainage; by puncture and washing of the sinuses.

A thorough endocrine study was made of most of the 38 patients who presented an overweight of more than 20 per cent. For this I am much indebted to Dr. Joseph H. Kaplan, head of the Medical Department of the Boston Dispensary. With a very slight incidence of minor pituitary disorders, the only outstanding feature in this study was the findings that obesity was present along with dysmenorrheal symptoms introductory to the menopause. In several instances the nasal symptoms leading to sinus disorders started at the same period as these irregularities; in three cases simultaneously with menopause of operative origin. Eight of the women among the 38 patients with overweight of over 20 per cent had between five and 12 children.

The *basal metabolic rate* has shown no definite trend and no deductions could be drawn from this source.

Of outstanding importance was the collaboration generously granted by the Food Clinic under the direction of Miss Frances Stern. In the perusal of the histories it was found that a great number of the patients had been under supervision of this department for a long period. With obesity as the diagnosis of admission, many of the patients had a long, carefully elaborated history in the records of the department. Much valuable information was gathered from these charts with their data regarding, among other things, environmental factors as the basis for establishing the food habits of the individual. It could be demonstrated that the complaints concerning overweight and sinus disorders have run together for long years, and many patients regard this connection as obvious. Trying to trace a food allergy was unsuccessful, even when some of the food groups were excluded from the diet for a long period; parallelism between fluctuation in body weight and nasal symptoms was found sometimes; a general weight loss was recorded and influenced the nasal complaints beneficially. No importance could be attached to any single food group partly because of the limited authenticity of the patient's subjective reports of his diet in many instances.

Analysis of food habits as to special national peculiarities or personal preferences offered little explanation. Thus it was planned to take refuge in the cardinal experiment; namely, to try to influence the nasal symptoms of the patient along with local treatment by reducing the body weight.

But it was found that in most cases such attempts were already made in the long course of dietary treatment. It was remarkable how often these attempts were frustrated by the attitude of these patients, who seemed to cling to their obesity and avoided dieting by offering the most variable excuses. Inadequate food money was often mentioned. Among other excuses were the following: the patient does not like vegetables and fruit; cannot resist taking sugar and sweet drinks; cannot resist eating extra bread and fruit; dieting causes nervousness; has not kept diet because of abscessed teeth; tried to be careful about diet but found it difficult during Passover; eats out often and as a guest cannot choose the food; follows the prescribed diet only from time to time, because of nervous stomach; cannot reduce because it is too cold, will do it in summer by sweating; finally confessing true

bulimia symptoms: would be pleased to lose weight but is always hungry.

Sometimes the mental ability was insufficient to understand the food requirements or why certain foods should be eaten and others should not. Many instances represented a long struggle on the part of the Food Clinic to induce the reluctant patient to change his or her eating habits for a reasonable diet.

It is obvious from the table including the participation of the five most numerous national groups, so compiled in the percentage of all patients frequenting the Boston Dispensary, that the individuals discussed in this report showed a higher incidence in three groups, computed for the patients with more than 20 per cent overweight; the Italian: +3.2 per cent; the Canadian: +9.6 per cent; the Jewish: +10.5 per cent.

COMMENT.

"It is generally accepted that something more than pure bacterial infection is required to produce sinus disease." This is the remark of Lederer[14] in his textbook where he emphasizes that in cases of chronicity, systemic factors, as allergic states, endocrine and diet imbalance should be investigated. As stated above, allergy played an insignificant rôle.

In our aim to distinguish between coincidence and true relationship several factors are to be taken into consideration. The first is the bearing of recurrent respiratory infection on sinus disease and vice versa. In the introductory remarks there is mentioned the relation of type of food to these infections. Adams[1] found the individual more subject to respiratory infection as a result of improper selection of food. "Shortness of breath is common, for the circulatory system, designed for a body weighing 150 pounds, is incapable of coping with a burden of 200 or 220 pounds." These patients offer a poor operative risk; many companies either refuse to insure or will insure only at higher premiums persons whom they rate as obese. Barlow[2] seems to put the two conditions in a parallel when he claims that in hypertrophic forms of arthritis the therapy should include removal of infectious foci *and* weight reduction. Obesity is always a danger

in acute infectious diseases such as pneumonia. Preble,[22] in his 1,000 patients with overweight (among them 300 from the Boston Dispensary), found obesity a serious condition conducive to heart, arterial disease and hypertension.

Levine[15] writes: "Obesity itself . . can produce shortness of breath. The stout person cannot breathe as freely as the lean. The diaphragm does not descend as readily and when the adiposity particularly involves the abdominal region the diaphragm is apt to be held in a high position and the vital capacity of the lung is diminished." Impairment of the pulmonary ventilation may be followed by chronic bronchitis and emphysema.

These general considerations will be even better understood when we include in the examination of the obese individual a careful evaluation of the state of his upper airways. One may quote as an example the connection with another special field of medicine; namely, orthopedics. It is routine to take care of associated mechanical complaints of obese people, from backaches to a breakdown in the arches of the foot.

In evaluation of the connection between obesity and upper respiratory infection much information can be obtained by studying the way in which Joslin[13] followed up the relation obesity-diabetes. Obesity acts as an exciting factor to produce the diabetes: As a parallel we stressed above the simultaneous appearance of overweight and sinus complaint. "So dangerous is obesity that acquiring diabetes, losing weight and coming under medical supervision may prolong the fat man's life." Remembering how frequently the complaint about sinuses induced us to ask for a general medical control, especially concerning overweight, we felt that this, too, would be of a definite general benefit for the patient. This may go as far as to be the decisive factor in the expectation of age between the thin and fat person.

The obvious objection that climate is the main source of sinus disorders, because they accumulate during the cold season, can be met in the following way: They are merely brought more in evidence by acute colds; they are by no means confined to the winter but present themselves with the swimming season and with periods of meteorologic depressions. In addition, the course of the disease over long periods

of years testifies that the warmer season produces at best only a lull in the manifestation of the symptoms. One may claim, just as for diabetes so for sinus infections, that obese persons who become affected with these conditions appear to be those with a susceptibility to the disease, the obesity acting as the exciting factor. Here, too, it is possible that the connection between the two conditions is not a causal one and that both reflect some underlying imbalance in the functioning of the body.

Registering the basal metabolic rate did not offer any special help. This is better understood when we know that Means[17] studied a series of 41 obese patients at the Massachusetts General Hospital and found an average variation from the expected metabolism of but 0.5 per cent.

Endocrinology and heredity do not offer the solution in our cases. Concerning the first, Adams[1] says: "We have no definite proof at present that the weight of presumably normal persons is to any extent influenced by glandular disorders." Concerning the second, Osler[20] can be quoted: "Sometimes the eating habits of the family may be responsible rather than any factor of heredity." "In the final analysis the most frequent cause of obesity is excess in food intake, especially of carbohydrates."

A conspicuous fact is the overwhelming majority of females, in number as well as in high percentages of excess weight. Whereas women number only 12 per cent more than men among all the patients of the Boston Dispensary, they number 84 among our 100 sinus patients, 61 of those overweight with an average of 24.2 per cent. The highest weight excess found in a man was 21.6 per cent; but not less than 30 women presented more than this, with the greatest weight excess being 92.1 per cent. In a small number menopausal factors may have been responsible for this remarkable ratio, but despite this it is not easy to explain why the bulk of the patients, 62 out of 100, were women between 31 and 60 years of age. Of the 38 patients with more than 20 per cent overweight, 34 were women, with an average age of 43.9 years. The preponderance of women between 30 and 60 years of age among the obese is well known, but as to etiology is as little understood, that Preble[23] had to take refuge in the following explanation: in the child-bearing period obstetricians and

pediatricians consider it necessary to "feed up" the ante-and postpartum patient without due regard to the general well-being of the patient. — It seems as if the trinity: "fat, female, forty," would indicate, as it is claimed for gall stones, also an inclination toward sinus disorders.

Remarkable is the low average height of our patients in this group of 100. This circumstance should call the attention in future investigations to the anthropometric factor.

Our patients, even when originating from lower economical classes, were far from being so poor as to lack food. Very characteristic is the repeatedly noted complaint that scarce food money is responsible for the inability to diet; the patient was compelled to eat normal food, which is easily available in the area in question, and besides being of faultless quality as a consequence of strictest control by the authorities, is very substantial and extraordinarily rich in carbohydrates. Easy accessibility of food rich in calories results in overnutrition. On the other hand, as regards abortive attempts to reduce by diet, so often mentioned in our case histories, it is known that abandoning the prescribed diet may bring the patient even beyond the previous weight.

The question arises as to whether it is justifiable to attribute the high incidence of sinus disease in the New Englanders to climatic factors as is customary; or should not rather a tendency to overweight be taken into consideration. Regarding this occurrence, two authorities may be quoted. Christian,[5] of Boston, in his revision of Osler's Principles (ed. 1938), writes on obesity: "The condition is increasing, particularly in the United States, where one sees an extraordinary number of very stout children." The regional circle is then drawn much closer by another eminent Bostonian: Joslin[13] studied a country town of New England and writes: . . the well-to-do were unusually fat. Eight per cent of 1,000 successive diabetics were found in 15 per cent below and 77 per cent above the standard weight zone. ". . People are fat because they eat too much. But this truth most of them hate to face. They take refuge in the fond illusion that heredity or glandular disturbance is to blame for their sad state." This statement receives a striking confirmation by the remarks of excuses as gathered above from the records of our Food

Clinic. Attempts to influence beneficially the upper respiratory condition by reducing met with the unexpected resistance to losing weight at all. Some patients were "staunch adherents of the doctrine that to be fat is to be healthy." As Means[17] said: "No one can be satisfactorily reduced who does not wish to be."

At this point the problem transcends the strictly somatic field. As transitional types, one may consider the neurotics among our patients. An underlying neurotic condition may lead to obesity and by way of nasal neuroses to disorders in this region. Adams[1] supposes that the fact that one person gains weight easily, the other with difficulty, finds its most plausible explanation in differences of temperament, of emotional stability. The urge to take care of his general systemic background helped in some cases to discover even more serious mental disorders. It is unjustifiable to discard the question referring simply to the proverbially slow cerebration of the obese. I am much obliged to Dr. Therese Benedek,[3] from the Psychoanalytical Institute of Chicago, for her permission to read the analysis of a bulimia case under publication. Her patient said, "I have orgies of eating, even if I loathe eating because I eat until it hurts."

Bruch,[4] in her extensive studies concerning obese children and their family frame, found cases where a rapid increase in weight occurred after some upsetting event in the family circle. In certain surroundings food has gained an exaggerated importance and stood for love, security and satisfaction. In the words of Newburgh:[19] "The obese are hyperemotional and use food as some people use tobacco or alcohol."

It is possible that the analytical approach would help to uncover a common neurotic basis for the conditions discussed in this paper. But while individual analyses in serials would be hardly practicable, less obstacles may present themselves with the class treatment as described by J. H. Pratt.[21] Made up of widely different races and different sects, these classes have a common bond in a common disease. Emerson formed his first class for undernourished children at the Boston Dispensary in 1908; the child who had made the greatest gain for the week sat in the place of honor. When a member of the class attained the proper weight for his height, he was "graduated" from the class and given a diploma. In the

tuberculosis class of Pratt the "graduates" dropped in at the meeting to get weighed; the greatest gains in weight were posted conspicuously each week on the blackboard. He quotes Mrs. Jacobson, a social worker of the Massachusetts General Hospital, regarding the contrary endeavor; that is, reducing: "Obese patients are difficult at first but once interested, many of them enjoy watching their weight with other patients in their class — the game being to lose a pound a week until normal weight is reached." In J. H. Pratt's[22] opinion, it is essential to have the help of social workers well trained in this special task. This would be one way to obtain the co-operation of these patients of "difficult" personality, and advance the knowledge concerning this psychosomatic problem.

To gain further information, it is planned to carry the investigation into the home of the patient. Data should be gathered about heredity, housing, food habits, sources of allergy, conjugal and parental relations. All this may help to uncover a common basis for the two conditions and promote a deeper understanding of their connection.

SUMMARY.

Study of 100 successive adult patients with sinus complaints resulted in finding a high incidence of cases with considerable overweight; especially in females between 30 and 60 years of age. Coexistence of upper airway infections and obesity could be traced back in many instances over a long period of time. No outstanding general or endocrine factor could be discovered and no connection with any particular food component. There is evidence that sinus disorders are caused by regional trends of the population to develop overweight as much as by the generally assumed climatic factor.

Attempts to influence the condition by reducing the body weight encountered a peculiar resistance of emotional-neurotic basis in these patients who found all kinds of excuses for neglecting or even rejecting prescribed diets.

Obesity seems to be a preconditioning factor in infections of the upper respiratory tract. On the other hand, obesity is in many cases the result of a neurotic trend for overeating, as had been demonstrated previously by others as well as by our own material.

This amounts to the following etiological pattern: neurotic eating habits lead to obesity, which facilitates sinus infection due to a decompensation in the function of the upper airways, because the circulatory and respiratory apparatus, established for a certain reasonable weight, is unable to cope with the new situation, represented by considerable overweight. If this etiological formula is correct, these cases of sinusitis may be considered not directly as a psychosomatic condition but as the secondary result of a psychosomatic condition; namely, obesity.

Continued clinical, environmental and analytical follow-up studies will be needed to decide whether one is justified in accepting a closer correlation between the two conditions, sinusitis and obesity.

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414 Beacon Street.

IN MEMORIAM

THOMAS JEFFERSON HARRIS, M.D.,

1866-1943.

Thomas J. Harris, a member of the American Otological Society for 40 years, died on March 14, 1943. Dr. Harris was born and spent his youth in Claremont, N. H. He was a descendant of Walter Harris, who came to America from England in 1632, and on the maternal side of Daniel Gookin, who also came from England in 1620. He was graduated from Dartmouth College, receiving his A.B. in 1886 and his A.M. in 1889. He received his M.D. from the University of Pennsylvania and then studied in Berlin and Vienna.

Returning to the United States, Dr. Harris served on the staffs of the New York Post-Graduate Hospital and the Manhattan Eye, Ear and Throat Hospital. Dr. Harris, a Lieutenant-Colonel during the first World War, was in charge of the Department of Otolaryngology, Geenral Hospital No. 14, Fort Oglethorpe, Ga. In addition he was Director of the School of Otolaryngology at Camp Greenleaf, Ga. He had numerous other hospital affiliations, one of which was Professor of Diseases of Nose and Throat at the New York Post-Graduate Medical School.

He served the American Otological Society long and faithfully, since his election to membership in 1903. He was its President during 1925-1926 and its Secretary for 20 years. In addition, he was a member and an officer in many medical, church and philanthropic organizations.

Dr. Harris wrote extensively on all phases of his specialty. His numerous contributions plus his personal efforts given without stint to the many medical and nonmedical groups will perpetuate his memory.

He leaves his widow, Mrs. Elizabeth Breed Harris, of Tucson, Ariz., and his daughter, Mrs. Elizabeth Barton, of Cambridge, Mass., to whom we extend our sincere sympathies.

CLIFTON MEREDITH MILLER, M.D.,

1874-1943.

Dr. Clifton M. Miller, prominent otolaryngologist of the South, died at his home in Richmond, Va., Feb. 26, after several months' illness.

Dr. Miller received his M.D. degree from the Medical College of Virginia in 1892, and his post-graduate work was done at the New York Eye and Ear Infirmary and St. Bartholomew's Clinic.

Aside from his busy practice, Dr. Miller was quite active in the civic, political and social life of Richmond.

He was Professor of Otology and Rhinology at the Medical College of Virginia, and was a Fellow of the American College of Surgeons, the American Otological Society, the American Laryngological, Rhinological and Otological Society and a member of its Council in 1933-1935.

Surviving him are his son, Clifton M. Miller, Jr., three daughters, the Misses Mary Bell and Emma Miller and Mrs. Murray Massie; two brothers, A. S. Miller, of Princeton, N. J., and Carroll Miller, of Washington, and two sisters, Mrs. Jesse Reeve, of Washington, and Mrs. Frank Schell, of Richmond, to all of whom we extend our sincere sympathy.